Preparedness and classical conditioning of fear : a critical inquiry

PREPAREDNESS AND CLASSICAL CONDITIONING OF FEAR:
A CRITICAL INQUIRY

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"There are more things in the workings of evolution than are dreamt of in the naive scenario of an advantageous allele sweeping to victory".

Kitcher (1985, p. 50).

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PREFACE

According to Marks (1988), experimental psychopathology is on its way to becoming a mature science. He maintains that a marked emphasis on Darwinian algorithms plays a decisive role in the rapidly expanding frontiers of psychopathological knowledge. Marks refers to the research on preparedness and phobias as a telling illustration of the fruitful conversation between evolutionary biology and psychopathology. Indeed, it has become customary to designate the preparedness hypothesis as a celebrated example of the way in which a biological approach can refine learning models of abnormal behavior (e.g., Eysenck, 1982; Mineka, 1986; Sturgis & Scott, 1984).

To the casual observer, the enormous growth of research into prepared conditioning in the past decade may seem to justify Marks' optimism about the scientific status of preparedness research. Yet, little more than a cursory glance at the literature on the subject will be sufficient to become aware of the theoretical flaws and empirical problems that seriously trouble the research on preparedness.

In the light of the current widespread belief that preparedness is a useful concept for theorizing about the origins of phobias, a critical examination of the preparedness hypothesis is not a luxury. In passing, it is worth noting that only two of the ten Anglo-Saxon reviews [1] that have appeared so far took issue on the dubious assumptions (Delprato, 1980) and the weak empirical basis (McNally, 1987) on which the preparedness hypothesis rests.

The primary purpose of the present dissertation is to comment on the theoretical and empirical inadequacies of the preparedness hypothesis. The basic thesis put forward here is that after having enjoyed a short period of progress, preparedness research is now in a state of complete stagnation. It will be argued that the number of straightforward failures and disappointments in preparedness research almost outweighs the number of explanatory successes.

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[1] Delprato (1980), McNally (1987), Öhman, Fredrikson & Hugdahl (1978a), Öhman (1979), Öhman & Hugdahl (1979), Öhman & Dimberg (1984), Öhman, Dimberg & Öst (1985), Öhman (1986), Öhman (1987), Rachman (1978).

A critical examination of an evolutionarily oriented hypothesis should be cautious in one particular respect. Claims about genetically determined behavior easily evoke ideological resistance (van den Berghe, 1982; Eibl-Eibesfeldt, 1979). The dispute about sociobiology exemplifies that when evolutionary hypotheses are involved, discussions between advocates and skeptics may degenerate into obscure rows. The lesson to be learned from this is that a critical examination of an evolutionary hypothesis should be limited to a review of the theoretical assumptions and the empirical evidence.

In the late fall of 1985, the present author set out to perform a series of studies concerned with preparedness and phobias. At that stage, it was generally believed that the major predictions of the preparedness hypothesis were covered by the empirical work by Öhman and co-workers (e.g., Mineka, 1985). However, the first attempt to replicate this work ended up with disappointing results (section 4.5). At first, these results seemed to represent an isolated anomaly. Indeed, the present author was close to throwing this failed replication onto the garbage heap of unpublished data when, in 1986, several papers from outside the Öhman group appeared in which unsuccessful replications (e.g., McNally & Foa, 1986) were reported. All in all, these studies pointed out the necessity of a critical re-analysis of the preparedness hypothesis. The following chapters reflect this course of events. Thus, the first three chapters briefly outline the main points of the preparedness hypothesis and the empirical evidence in favor of this hypothesis. Chapter 4 consists of a set of separate studies, none of which was able to confirm the main predictions of the preparedness hypothesis. In the next chapter, a number of theoretical inadequacies of this hypothesis are highlighted. Chapter 6 attempts to critically evaluate the preparedness concept with a concern for "the facts of the matter". In the final chapter a case is made out for the usefulness of neuroscientific knowledge in redefining the classical conditioning model of phobias.

CHAPTER ONE

THEORETICAL BACKGROUND OF THE PREPAREDNESS HYPOTHESIS[1]

It is a truism that new scientific concepts are invoked to resolve theoretical problems. Seligman's (1970, 1971) preparedness hypothesis is no exception to this rule. It can best be understood as an attempt to revise the conditioning model of phobias by adding Darwinian notions to this model. However, for appreciating the form that this revision took, the main traditions that dominated experimental psychology in the early seventies have to be taken into account. It is beyond doubt that what is generally referred to as "the cognitive revolution" (Dember, 1974) resulted in many researchers "going cognitive". Yet, elsewhere a climate began to develop in which biological concepts were preferred to cognitive theorizing. Seligman's preparedness hypothesis fitted in nicely with such a climate. Apart from this, two discrete events, one of a personal and the other one of a scientific nature, strongly inspired Seligman in his formulation of the preparedness concept.

The present chapter will be an attempt to elucidate the theoretical background of Seligman's hypothesis. The first part will indicate the shortcomings of the traditional conditioning model of phobias. When these shortcomings became increasingly clear in the late sixties, they gave rise to an urgent need for new concepts such as, for example, "preparedness". The second part will be concerned with the growing popularity of evolutionary notions in the early seventies. The third and final part of this chapter will describe two events which suggested to Seligman that evolutionary concepts might be successfully applied to conditioning phenomena in general and to phobias in particular.

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[1]This chapter is partly based on several articles (Merckelbach & Jansen, 1986; Merckelbach, 1986; Jansen & Merckelbach, 1988) which appeared earlier in Dutch journals. Publication facts of articles by the present author that are referred to in the text can be found in the "List of publications and abstracts".

1.1 After Watson

Ever since Watson and Rayner (1920) published their "little Albert" study, psychologists have tried time and again to condition and decondition human fear reactions to a neutral stimulus (conditioned stimulus; CS) by pairing that stimulus with an aversive event (unconditioned stimulus; UCS). Some attempts at this failed (Bregman, 1934), others succeeded (M.C. Jones, 1924; H.E. Jones, 1930), and still others yielded mixed results (English, 1929; Valentine, 1930). While failures to document fear conditioning attracted little attention, the successes were taken as strong support of the conditioning model of phobias (e.g., Eysenck & Rachman, 1965), which implies that

> "anxiety (fear) is the conditioned form of the pain reaction" (Mowrer, 1939; p. 555).

In fact, they gave rise to a state of euphoria in which it was generally believed that experimentally manipulated associations between arbitrary stimuli can imitate the etiology and the course of phobic fears (Eysenck, 1982). The following quotations are illustrative:

> "The general rule is that any stimulus may become a danger signal provided it is immediately and repeatedly followed with any injurious or painful experience" (May, 1944; p. 72).

> "Any 'neutral' stimulus, simple or complex, that happens to make an impact on an individual at about the time that a fear reaction is evoked acquires the ability to evoke fear subsequently" (Wolpe & Rachman, 1960; p. 145).

This was also the context in which accounts that overestimated and, ultimately, distorted the significance of the "little Albert" study became very common (Harris, 1979; Samelson, 1980).

The optimism about the capability of the conditioning model to come to grips with the origins of phobias gradually made way for the awareness that this model failed in many respects. A number of researchers

(Eysenck, 1968, 1976, 1979, 1982; Gossop, 1981; Gray, 1982; Marks, 1977; 1987; Rachman, 1976, 1977) pointed out the following shortcomings of the conditioning model.

First, it is a well-established epidemiological (Costello, 1982) and clinical (Marks, 1969) fact that fears are directed at a selective and non-arbitrary set of objects and situations . From a Watsonian point of view this non-random distribution of phobias is hard to understand. That is, on the basis of the conditioning model, it is expected that each signal can become a phobic cue, provided that it is followed by an aversive event. This is obviously not the case. Agras, Sylvester, and Oliveau (1969), for example, reported that in a normal population, fear of snakes occurs almost twice as often as fear of dentists.

Second, in the literature excellent illustrations can be found of intensely terrifying situations (e.g., air raids, sieges) in which, contrary to all predictions deriving from the conditioning model, people did not acquire phobic fears (Rachman, 1977; Saigh, 1984).

Third, over the years it has become clear that not all phobias can be traced back to a confrontation with (sub)-traumatic or aversive events (Marks, 1969; Goorney & O'Connor, 1971; Wolpe, Lande, McNally & Schotte, 1985). There is good reason to consider other pathways to fear such as vicarious learning and information transmission (Rachman, 1977).

Fourth, laboratory conditioning of fear indices is dependent on highly refined experimental conditions (e.g., the temporal relations between CS and UCS), requires multiple CS-UCS trials rather than a single trial procedure, and, eventually, results in a conditioned response that quickly recedes (Eysenck, 1979). Consequently, what can be accomplished in a laboratory does not at all resemble the etiology and course of real life phobias.

Fifth, the conditioning model is unable to explain in a straightforward manner why some phobias are diagnosed more often in women than in men, why the onset of, for example, social phobias and agoraphobias predominantly occurs in early adult life (Gray, 1982), and why phobias often do not only fail to extinguish but even increase in strength in the absence of an observable UCS (Eysenck, 1968).

In view of the aforementioned points, investigators and clinicians alike generally concluded that a conditioning model of the Watsonian type

was no longer defensible (e.g., Eysenck, 1979; Marks, 1977). Yet, ideas about the way in which theoretical progress should be achieved differed markedly. On the one hand, there was a number of authors (Mackay, 1983; Beck, Emery & Greenberg, 1985) who felt that the conditioning model was definitely and in all respects untenable and that more cognitively oriented theories had to replace it. On the other hand, attempts were made to revise and update the conditioning model by introducing new concepts, such as "incubation" (Eysenck, 1968; Eysenck, 1987a) and "preparedness" (Seligman, 1970; 1988). A remarkable feature of these attempts was that they emphasized biological rather than cognitive factors.

1.2 Darwin revisited

Referring to the role of Darwinian notions in psychology, Ghiselin writes, as late as 1973:

> "The history of the assimilation of Darwin is the history of the failure to assimilate Darwinism. But Darwin's contribution to psychology was neither understood nor accepted, and only now are we beginning to realize what that contribution was" (p. 968).

The 1970s were, indeed, characterized by a simultaneous downfall of extreme environmentalism and an influx of evolutionary and ethological concepts into academic psychology (Delprato, 1987). In 1971, Razran published his "Mind in evolution" which sought to reconcile Pavlovian and Darwinian notions. At about the same time, students of emotions (e.g., Ekman, 1973; Izard, 1971) began to rediscover the relevance of Darwin's (1872, 1965) "Expression of emotions in man and animals". The role of evolutionary mechanisms in social behavior was underlined in programmatic texts, such as Jürgens and Ploog's (1974) "Von der Ethologie zur Psychologie", Wilson's (1975) "Sociobiology: The new synthesis", and Eibl-Eibesfeld's (1979) "Human ethology: concepts and implications for the sciences of man".

The rising tide of evolutionary considerations within psychology

led many investigators to taking issue on the "general-process view of learning" which dominated early behaviorism (Schwartz, 1974; Herrnstein, 1977, Johnston, 1981). According to this view, the study of arbitrary reinforcers, operants, and stimuli would yield a small set of general principles (e.g., the law of effect and the law of contiguity) that underlie all manifestations of learning. However, starting with Breland and Breland's (1961) paper "The misbehavior of organisms", reports of learning biases which were difficult to capture in terms of the general-process view began to appear. Breland and Breland observed, for example, that when raccoons are reinforced for picking up coins, the response gradually gives way to coinwashing behavior. They assumed that an "instinctive drive" infiltrated and, ultimately, overrode the reinforced response. Another instance of a learning bias was provided by Bolles (1971), who reported that rats learn an avoidance response which consists of running or jumping away more readily than one which consists of pressing a bar. He argued that the strength of avoidance learning is highly dependent on the extent to which the pre-selected avoidance response is related to species-specific defense reactions. Similarly, Shettleworth (1975) pointed out cases in which species-specific feeding behavior affected operant conditioning. He showed that in hamsters the frequency of digging can be enhanced by food reinforcers but that of face-washing cannot. What these examples suggest, then, is that operant learning is heavily biased by the biological features of the organism under consideration. As will be described below, the Garcia effect illustrates that Pavlovian learning is equally prone to this type of bias.

As the number of clearly documented learning biases grew, the general-process view of learning gradually made way for a "heredity x environment movement", as Delprato (1987) called it. Central to this movement was the idea that operant and Pavlovian principles are confined by biological boundaries rather than that they apply equally well to all possible combinations of reinforcers, stimuli, and responses.

As far as psychopathology is concerned, it should be noted that phobias as the outcome of evolutionarily biased conditioning was Seligman's new formulation but, essentially, not his new idea. With this formulation Seligman found himself in the company of the early psycho-

analysts whose writings offer many speculations about an ancient component in neurotic disorders (Sulloway, 1979). The following quotations are from Freud's (1917, 1974) " Introductory lectures on psychoanalysis".

> "I have repeatedly been led to suspect that the psychology of the neuroses has stored up in it more of the antiquities of human development than any other source" (p. 418).

> "It must be admitted, subject to the necessary qualifications, that among the contents of phobias there are a number which, as Stanley Hall insists, are adopted to serve as objects of anxiety owing to phylogenetic inheritance" (p. 460).

In contrast with this, Watson (1924), one of the founding fathers of behaviorism and behavior therapy, fiercly rejected the evolutionary or genetic dimension of behavior:

> "We draw the conclusion that there is no such thing as an inheritance of capacity, talent, temperament, mental constitution, and characteristics. These things again depend on training that goes on mainly in the cradle" (p. 93).

Eysenck (1982, 1987b) argued that the ignorance of biological factors which characterized behaviorism and, in its wake, behavior therapy, can be traced back to Watson's extreme environmentalism (but see also Delprato, 1987). On the other hand, Razran (1971) held authors such as Skinner and Hull responsible for this state of affairs (but see also Herrnstein, 1977). Be that as it may, in accordance with a general re-evaluation of biological and ethological models, this ignorance no longer dominated the behavioral study of neurotic behavior in the 1970s. Apart from Seligman's (1971) preparedness concept, the following hypotheses and speculations are indicative of a shift towards evolutionary biology:

1. Holland's (1974) hypothesis that compulsive behavior is related to displacement activities typically seen in threatened animals.

2. Marks' (1976) hypothesis about the parallels between death-feigning and tonic immobility observed in fearful animals and the vaso-vagal reflex commonly found in blood phobics.
3. McGuire's (1986; McGuire & Essock-Vitale, 1982) speculations about the evolutionary advantage of the ability to imagine and its relation to the heightened frequency of unrealistic wishes in neurotic depression.

1.3 Sauce Béarnaise, the Garcia effect, and the prepared character of phobias

In his 1970 article, Seligman pointed out that much research concerned with aversive classical conditioning is governed by the assumption that, once they are paired with a UCS, all types of CSs are equally capable of eliciting a conditioned response (CR). Referring to this assumption as the premise of equivalence or equipotentiality (Seligman & Hager, 1972), Seligman went on to argue that this premise is untenable and has to be replaced by the concept of preparedness. In its simplest form, the concept of preparedness imports that the ease with which CS-UCS associations evoke a CR is affected by the evolutionary background of the organism. A more detailed description of the preparedness concept will be given hereafter. For the time being, it suffices to remark that this concept differs radically from the premise of equipotentiality by stressing the non-equivalence of stimuli due to their varying degree of evolutionary significance. Thus, the preparedness concept is a straightforward example of a "heredity x environment" hypothesis.

According to Seligman's own reconstruction (Seligman & Hager, 1972), a personal experience and a scientific discovery played a major role in the formulation of the preparedness concept. As regards the personal event, Seligman (Seligman & Hager, 1972) reports that one day he and his wife ate his favorite Sauce Béarnaise and afterwards listened to Wagner's Tristan and Isolde. That same night he got sick and spent most of the time vomiting. Despite the fact that he later learned that his illness was caused by a stomach flu, he developed a persistent aversion to Sauce Béarnaise. Moreover, the aversion was specifically focussed on this food item and incorporated neither his wife nor Wagner's opera. The selectivity with which the aversion was directed at the food as well as the

interval of six hours between the food intake and the vomiting made an explanation of the Sauce Béarnaise aversion based on classical conditioning, - with Sauce Béarnaise as the CS, illness as the UCS, vomiting as the unconditioned response (UCR), and aversion as the CR -, implausible at first glance.

The Sauce Béarnaise incident nearly coincided with the first publications on the selectivity of taste aversion (see review by Garcia, McGowan & Green, 1972). In what would become a well-known experiment, Garcia and Koelling (1966) exposed two groups of water-deprived rats to a compound stimulus ("bright-noisy, saccharin-tasting water") consisting of an audiovisual signal and flavored water. One group was punished by electric shocks to the paws in the presence of the compound stimulus and the other group was punished by a toxin injection inducing nausea. During a subsequent extinction test the capability of the separate component stimuli to deter the rats from drinking was examined. It was found that in the toxin group only the taste cue became a CS for suppression of water intake, whereas in the shock group only the audiovisual signal became a CS for suppression. Obviously, rats associate toxin induced illness more readily with taste cues than with audiovisual signals, a phenomenon which later became known as the Garcia effect (e.g., Eelen, 1982). Although similar results were obtained in slightly different experimental set-ups (Garcia & Koelling, 1967; Garcia, McGowan, Ervin & Koelling, 1968), the article based upon the experiment described above was rejected by some illustrious journals.

The parallel between the Sauce Béarnaise incident and the Garcia effect became apparent when, a few months later, Garcia, Ervin, and Koelling (1966) published a second article. In this article the authors reported that rats acquired an aversion to saccharin-flavored water even when a nausea inducing apomorphine injection was given 22 minutes after drinking. Again, the research by Garcia and co-workers received a cool reception, which is exemplified by the following remark:

> "Those findings are no more likely than birdshit in a cuckoo clock" (cited in Seligman & Hager, 1972; p. 15).

Seligman (1970) and Garcia, McGowan, and Green (1972) reasoned that the Sauce Béarnaise and Garcia effects can best be understood as evolutionarily biased learning. In other words, the readiness or preparedness of organisms to associate taste cues (CS) with illness (UCS), even when there is a considerable time gap between these stimuli, promotes the avoidance of potentially toxic food and, hence, increases survival chances. Thus, the Sauce Béarnaise and Garcia phenomena were presented as instances in which Pavlovian conditioning was controlled by phylogenetic factors.

In 1971, Seligman drew attention to the possible role of preparedness in the etiology of phobic fears by arguing that what is true of taste aversion might also hold for phobias. According to Seligman, most phobic fears result from evolutionarily prepared conditioning. In this form of conditioning, fear is readily associated with precisely those objects and situations that used to be threatening to prehistoric man. Hence, it deviates from the typical laboratory situation in which fear indices are conditioned by means of stimuli chosen arbitrarily (e.g., tones and shocks).

CHAPTER TWO

EVOLUTION AND PHOBIAS[1]

The idea that the conditioning model of phobias could benefit from insights of evolutionary biology at first glance seems odd. Assuming that phobic behavior is maladaptive, it is hard to see why the principle of natural selection would have favored such a behavior. Two approaches characterize earlier discussions of this subject (McGuire, 1986). The first approach proposes that some dysfunctions confer a selective advantage and are, consequently, not eliminated by selective pressure. The genetic dynamics behind sickle cell anemia are a case in point (e.g., Kitcher, 1985); the allele for sickle cell anemia is maintained in some African environments because it enhances resistance to malaria. The view that clinical depression may be adaptive in the sense that it elicits empathy and caretaking behavior (Lewis, 1934) is related to the "dysfunction as selective advantage" approach. Another example of this approach is provided by theories which argue that obsessive-compulsive behavior results in excessive cleanliness and, therefore, reduces vulnerability and maximizes fitness (e.g., Heisel, 1988).

The second approach assumes that some dysfunctions reflect the emergence of "atavistic" behavior that used to contribute to the organism's survival. It focusses on the desynchrony which is introduced by the inertia of the gene pool and a rapid ecological change. The purpose of the present chapter is to show that the hypothesis of phobias as evolutionarily prepared behavior follows this approach. The first section summarizes the main points of Seligman's (1971) version of this hypothesis. The second section describes Öhman's extension of the preparedness hypothesis.

2.1 Seligman's first two rungs

Kitcher (1985) refers to the sociobiological attempt (e.g., Wilson,

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[1]This chapter is partly based on Merckelbach, van den Hout, and van der Molen (1987a).

1978) to ascend from genes to human behavior as "Wilson's ladder". This ladder summarizes the flow of the sociobiological argumentation and consists of the following rungs:

1. It is assumed that under certain conditions certain forms of behavior maximize fitness.
2. If these forms of behavior appear to be widespread, it is concluded that they are the result of natural selection.
3. As natural selection acts on genes, it is proposed that these forms of behavior have a genetic basis.
4. Since these forms of behavior are genetically determined, they are thought to be insensitive to environmental manipulation.

The correspondence between the preparedness hypothesis and sociobiological reasoning expresses itself in Lumsden and Wilson's (1982) qualification of the preparedness hypothesis as a "dramatic illustration" of the way in which genes affect culture. Another indication of this correspondence is the article by Öhman and Dimberg (1984), in which sociobiological notions play an important part. Thus, **mutatis mutandis**, "Wilson's ladder" can be used for going through the basic propositions of the preparedness hypothesis systematically.

In turning now to Seligman (1971), it should be remembered that it was his intention to account for the fact that most phobias pertain to a relatively narrow class of stimuli. According to Seligman, a close inspection of this class reveals that it consists of objects and events which were probably threatening to prehistoric man (e.g., thunderstorms, predators, heights, etc.). Thus, Seligman arrives at the first rung by assuming that most phobias used to be adaptive in that they "facilitated survival and/or promoted differential reproduction of homo sapiens in pretechnological society" (De Silva, Rachman & Seligman, 1977; p. 67). He then proceeds to the second rung of "Wilson's ladder" by suggesting that natural selection has put an adaptive premium on those individuals who readily learned to avoid dangerous objects and events (Seligman & Hager, 1972). Phobias are thought to be akin to learning biases of the type documented by Garcia and Koelling (1966) in that both phenomena result from evolutionarily prepared learning, that is, learning that involves

contingencies which are or were survival relevant.

Seligman's (1970) operational definition of "preparedness" runs as follows:

> "The relative preparedness of an organism for learning about a situation is defined by the amount of input (e.g. number of trials, pairings, bits of information, etc.) which must occur before that output (responses, acts, repertoire, etc.), which is construed as evidence of acquisitions, reliably occurs (...). If the organism makes the indicated response consistently from the very first presentation of the CS on, such "learning" represents a clear case of instinctive responding, the extreme of the prepared end of the dimension. If the organism makes the response consistently after only a few pairings, it is somewhat prepared. If the response emerges only after very many pairings or does not occur at all, the organism is said to be contraprepared" (p. 408).

What is stressed in this definition is the continuum between instinct and learning. Both phobias and taste aversions are supposed to represent a type of learning that is modulated by instinct. Taking taste aversion as the prototype of prepared learning, Seligman (1971) attributes a number of qualities to this type of learning in general and to phobias in particular (see Table 1).

Table 1. CHARACTERISTICS OF "PREPARED" PHOBIAS.

Characteristic	Theoretical Status
Easy acquisition	Hypothetical; By analogy with taste aversion
Selectivity due to survival relevance	By definition; By analogy with taste aversion
Resistance to extinction	Through "biological" connotations of survival relevance
Insensitivity to instructions	Idem

More specifically, it is argued that prepared learning does occur, even with remote CS–UCS pairings or UCSs consisting of "slight provocations", that CSs represent survival-relevant cues, and, finally, that the resulting CRs are highly persistent and not easily modified by instructions. Applied to phobias this means: Phobias can be acquired in the absence of a traumatic UCS, they are mostly directed at cues which used to be survival relevant, they resist modification, and they are insensitive to rational argumentation.

With the last mentioned characteristics, - failure to extinguish and non-cognitivity -, Seligman (1971) comes close to the fourth rung of "Wilson's ladder". However, nowhere would one find Seligman to assert that phobias are directly connected with genes. Although a critical examination of Seligman's proposal is postponed until the later chapters, it should be recognized that a comprehensive summary of Seligman's hypothesis is difficult to arrive at, exactly because his argumentation is rather of a loose and evocative nature than logically conceived. That is to say, without theoretical considerations, Seligman qualifies prepared learning as "primitive" (Seligman & Hager, 1972; p. 114, p. 464), "blind" (idem; p. 97), and "robust" (idem; p. 97), probably because this learning is assumed to result from evolutionary pressure.

In sum, easy acquisition is regarded as the outstanding feature of prepared learning. The hypothesis of survival relevance is called in to account for this easy acquisition. Non-cognitivity and resistance to extinction are connotations of survival relevance, evolution, biology, etc..

Seligman and others (e.g., Rachman, 1977) have gone to great lengths to show that, compared to the "traditional" conditioning model, the preparedness hypothesis is better able to explain the typical features of phobias.

1. The selectivity of phobic cues is accounted for by assuming that these cues used to be threatening to our ancestors (Seligman, 1971).
2. Modern war-related stimuli are phylogenetically too recent to allow prepared learning to occur. "These modern artifacts do not feature in our biological inheritance" (Rachman, 1977; p. 384). This would explain why a number of terrifying situations (e.g., air raids) seldom give rise to an increase in phobic fears.

3. Although the preparedness hypothesis maintains that some CS-UCS association is required for phobias to occur, it also allows for the possibility that the UCS consists of a "slight provocation". Provided that the CS represents a "phylogenetic" danger, a mild UCS is sufficient to elicit phobic fears (Rachman & Seligman, 1976).
4. Prepared learning violates principles that the general-process view (section 1.2) considered to be essential to Pavlovian conditioning. That is, "prepared" phobias may occur as the result of a remote CS-UCS association. Moreover, "prepared" phobias are resistant to extinction by virtue of their evolutionary origins.

To summarize, given a "phylogenetically" dangerous object, say a snake, a single mildly aversive experience which occurs some time later is sufficient for fear conditioning to take place. The resulting fear is insensitive to rational argumentation and resistant to extinction. A case quoted by Marks (1977) exemplifies the etiology of "prepared phobias".

> "A four-year-old girl was playing in the park. Thinking that she saw a snake, she ran to her parents' car and jumped inside, slamming the door behind her. Unfortunately the girl's hand was caught by the closing car door, the results of which were severe pain and several visits to the doctor. Before this she may have been afraid of snakes, but not phobic. After this experience a phobia developed, not of cars or car doors, but of snakes. The snake phobia persisted into adulthood (....)" (p. 192).

2.2 The missing rung: Öhman's contribution to the preparedness hypothesis

Paraphrasing Eysenck (1987b), it can be argued that a discussion about the question whether genetic factors or learning factors determine behavior is as senseless as a debate about the question whether the length or the width of a field defines its area. However, this does not alter the fact that in the case of Seligman's preparedness hypothesis there is room for doubt when it comes to specifying the role of genetics. Indeed, close reading of the early papers on preparedness (e.g., Selig-

man, 1971; Rachman & Seligman, 1976; De Silva, Rachman & Seligman, 1977) leaves one with the uneasy feeling that Seligman's conceptualization is somehow of the Lamarckian type, because it skips over the genetic details so easily (section 5.1). Öhman and Dimberg (1984) must be given credit for their attempt to provide the preparedness hypothesis with a more coherent perspective on the interaction between learning and genes.

In order to elucidate the role of the genetic component in the etiology of phobias, Öhman and Dimberg (1984) distinguish between closed and open genetic programs. Drawing on a model introduced by Mayr (1974), they define closed genetic programs as programs in which genetic information automatically shapes neural structures and, consequently, behavior. In open genetic programs, the connection between genes and behavior is less direct. That is to say, for open genetic programs to become translated into neural programs, specific environmental input is required. Open genetic programs allow the organism to take advantage of individual learning experiences. According to Öhman and Dimberg, closed and open genetic programs differ in many respects (see Table 2). Evolution capitalizes on closed programs when the species faces a more or less constant environment.

Table 2. CHARACTERISTICS OF OPEN AND CLOSED GENETIC PROGRAMS.

	Closed Programs	Open Programs
Ecological niche	Stable over generations	Rapidly changing
Mode of selection	Natural selection	Reinforcement
Unit of selection	Organism **in toto**	Individual behavior
Type of selection	R-selection	K-selection
Type of organism	Lower animals	Higher mammals

In these programs, genetic features are fed back and retained in the common gene pool, provided that the types of behavior to which they give rise are evaluated positively by natural selection. However, natural selection operates on organisms **in toto** rather than on single adaptive behavior. Furthermore, closed genetic programs are related to an r-

selection strategy; many descendants with high death rate and low parental dependence are typical for the reproduction strategy associated with these programs.

In contrast, open genetic programs enable the organism to react in a flexible way to a rapidly changing environment. Behavior controlled by open genetic programs is primarily evaluated by reinforcement which acts on specific kinds of behavior rather than on the total organism. Open genetic programs follow a K-selection strategy. This strategy is characterized by few descendants, low death rate, and high parental investment.

When comparing open and closed programs, Öhman and Dimberg emphasize two points. First, they stress that it is not appropriate to think of open and closed programs as antithetical processes. Instead, open and closed programs refer to the respective ends of a continuum. Viewed from this perspective, phobic responses to survival-relevant cues (e.g., snakes, spiders, etc.) are controlled by relatively closed genetic programs, whereas driving, swimming, using statistics etc. - briefly, the general-process types of learning (Schwartz, 1974) - are controlled by relatively open genetic programs. Second, Öhman and Dimberg (1984) argue that even in relatively closed programs, learning is important. In phenomena such as imprinting, taste aversion and phobias, learning plays a decisive role to the extent that it is left to the organism's experience to define the precise features of the parent, toxic agent or predator respectively. Although learning is directly evaluated by reinforcement, it is also indirectly subjected to natural selection. That is to say, natural selection acts on learning capacity rather than on learning behavior itself.

> "Thus, individuals with a successful capacity for learning are likely to leave more offspring than individuals with less successful learning capacities. It is the capacity for learning, then, which becomes represented in the gene pool" (Öhman & Dimberg, 1984; p. 59/60).

To sum up, it may be stated that, in contrast to Seligman (1971), Öhman and Dimberg (1984) offer a theoretical framework for preparedness that is located on the third rung of Wilson's ladder (section 2.1). In

their formulation, phobias are the product of a relatively closed genetic program in which a little experience joins a genetically represented capacity to learn about dangerous things.

2.3 Making the black box white

The strict behaviorist's black box model relies on stimuli, responses, and a small set of general learning principles. It deliberately ignores organismic processes. It goes without saying that such a model is unable to deal with the selective stimulus-response associations that are involved in taste aversion and, possibly, in phobias. Broadly speaking, the preparedness hypothesis, and especially Öhman's version, can be characterized as an attempt to fill the black box with evolutionarily shaped mechanisms.

The general-process type of studies (section 1.2) concerned with conditioning and phobias assumed that fear is a "unitary" concept and that fear conditioned in a laboratory is an equivalent of clinical phobias (e.g., Solomon & Wynne, 1954; Campbell, Sanderson & Laverty, 1964; Geer, 1968). Öhman, Dimberg, and Öst (1985) and Öhman (1986) criticised these assumptions. The central point raised by these investigators is that the characteristics of fear vary as a function of the behavioral context in which the fear occurs. As for this behavioral context, Öhman et al. (1985) distinguish between noncommunicative, interspecific, and intraspecific behavior, thereby drawing on a descriptive model advanced by Mayr (1974). Noncommunicative behavior refers to behavior directed at the material aspects of the environment, interspecific behavior refers to a confrontation with animals of a different species, and intraspecific behavior refers to the communication between animals of the same species. Fears in the noncommunicative category correspond with phobias of inanimate objects (e.g., heights, enclosed places), whereas fears in the inter- an intraspecific categories correspond with animal and social phobias respectively. In their discussion, Öhman et al. (1985) concentrate on the last two categories.

A similarity between fear originating in the interspecific category and that originating in the intraspecific category is that both types of fear seem to be controlled by relatively closed genetic programs (Mayr,

1974; see, however, section 5.2). The potential differences between these two types of fear become clear when their biological functions are analyzed. Whereas interspecific fear stems from a functional system that is in charge of defense strategies against predators, intraspecific fear is related to a system which regulates social hierarchies (the social submissiveness system). The predatory defense system and the social submissiveness system are activated by completely different classes of stimuli. Whereas the former is mobilized by features which are likely to be associated with a predator, the latter is mobilized by threatening facial or postural expressions. As regards the perceptual triggers for the predatory defense system, Öhman et al. (1985) speculate about the prototypical role of reptilian stimuli:

> "Given that there were reptile predators of potential danger for many species around even after the demise of the dinosaurs, it is likely that genes favoring quick learning of avoidance and escape from reptilian stimuli were left in the gene pools of many mammals, unless the contrary was explicitly selected for. Furthermore, given the opportunism of evolution, it is not unlikely that similar defense strategies to new predator pressures were build around those originally designed for reptiles" (p. 20).

The predatory defense system and the social submissiveness system also differ as to their developmental courses. The predatory defense system is urgently needed as soon as a child is able to move away from its parent. The social submissiveness system, on the other hand, establishes social hierarchies during adolescence.

A final difference between the two systems concerns the risks involved in both systems. Organisms equipped with a badly functioning predatory defense system run the risk of ending up as a prey. In contrast with this, social conflicts seldom result in a deadly attack. Thus, the predatory defense system consists of automatic processing routines which enable the organism to react with a reflexive response to threat. Since it prepares the organism for action, the sympathetic branch of the nervous system plays an important role in these automatic routines. The

social submissiveness system is of a less automatic nature and allows controlled processing routines. Consequently, the role of the sympathetic nervous system is limited.

To sum up, compared with Seligman's position, Öhman et al. (1985) propose a more detailed, ethological description of the selectivity of fears. From this description they proceed to theorize about two evolutionarily based systems that operate within the realm of the black box. In a way, their **exposé** is useful as it broadens the explanatory power of the preparedness hypothesis. In addition to the characteristics that Seligman attributes to "prepared" phobias (see section 2.1), Öhman's formulation has two implications:

1. As the predatory defense system is mobilized in early childhood and the social submissiveness system is related to a later developmental phase, the age of onset of animal and social phobias should be quite different.
2. Compared to social phobias, animal phobias should be associated with more automatic processing and higher levels of sympathetic activity.

CHAPTER THREE

EMPIRICAL SUPPORT FOR THE PREPAREDNESS HYPOTHESIS[1]

To claim that a particular aspect of behavior is evolutionarily preprogrammed is one thing. To gather empirical evidence for such a claim is another and a more difficult task to accomplish. In the case of evolutionary hypotheses about human behavior, empirical problems lurk in every corner. Lewontin (1979) was probably right when he qualified most of these hypotheses as "imaginative reconstructions" which entirely rely on plausibility. Seligman and colleagues were certainly aware of this. They admitted that evolutionary arguments

> "are rather slippery and can be glibly made. The only way of validating the view that, say, avoiding going out in the dark has been selected for in evolution to a greater extent than avoiding eating in public places, would be to recreate past evolutionary pressures and see which phobia is more amplified over generations. Such an experiment verges on the impossible and is unlikely to be funded" (De Silva, Rachman & Seligman, 1977; p. 74).

As a result, the empirical work in this field has concentrated on the verification of the implications rather than on the basic tenets of the preparedness concept.

The present chapter will summarize the findings that lend support to the implications of the preparedness hypothesis. These findings stem from various sources. Section 3.1 will deal with fear-survey studies that documented the non-random distribution of human fears. Section 3.2 will go into the details of clinical studies which claimed to have shown that this non-random distribution reflects the "phylogenetic dangerousness" of feared stimuli. Additionally, it will draw attention to data that suggest that animal and social phobias are associated with different

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[1] This chapter is an extended version of Merckelbach, van den Hout, and van der Molen (1989).

onset ages and psychophysiological response patterns. Section 3.3 describes the illusory correlation paradigm. In connection with this paradigm it was found that normal subjects are prone to make biased estimates when confronted with "phylogenetically dangerous" stimuli. These stimuli also play a major role in the psychophysiological conditioning experiments and animal studies that are reviewed in section 3.4 and section 3.5 respectively.

3.1 What is prepared? Fear surveys among normal subjects

In anthropological literature, anecdotal cases can be found that testify to the strange, non-random distribution of fears. For instance, in a study of Canadian Indians, Hallowell (1938) reported that the Indians were fearless of wolves and bears, but terrified of frogs and toads. However, when the selectivity of fear is brought up for discussion, advocates of the preparedness hypothesis as a rule refer to two epidemiological surveys. The first study was carried out by Agras et al. (1969) and was based on a large sample of adults (N=325) living in a small American town. Among others things, the researchers found snake fears to be prevalent twice as often as dental fears. The second, more recent study (Costello, 1982) reported similar results. In this study, 449 women were selected at random from 10 communities in Alberta, Canada and were interviewed. It was concluded that animal fears (which included fear of dogs, snakes, cats, and/or spiders) were the most prevalent, followed by nature (e.g., heights), mutilation (e.g., injections), social, and separation (e.g., taking journeys) fears. In effect, animal fears occurred in nearly 43% of the women. What these two community studies demonstrate, therefore, is that fears are, indeed, distributed non-randomly. Furthermore, the finding that fears are far more frequently directed at "phylogenetically dangerous" stimuli (e.g., snakes) than at "ontogenetically dangerous" stimuli (e.g., injections) can be taken as indirect support of the preparedness concept.

It is possible, of course, to interpret the selectivity of fears in terms of culturally determined, negative connotations of animals such as snakes. Advocates of the preparedness hypothesis argue that this interpretation does not represent a serious obstacle to their hypothesis.

They suggest that the negative connotations of, for example, snakes are found among many cultures (Öhman et al., 1985). Such a universality would underline rather than disqualify the preparedness hypothesis. Thus, Öhman (1986) wonders whether

> "It is merely an accident that the embodiments of Evil often have bestial features. An alternative possibility is that those features have some referents in the collective information storage device known as the human gene pool, as implied by Jung's concept of archetypes. Examples of mythical versions of encounters with beasts abound. They include, for instance, Hercules' fight with the many headed hydra of Greek mythology, and Tor's wrestling with the "Midgards-snake" in Nordic Viking mythology" (p. 123).

Assuming that evolutionary forces participate in the etiology of, say, snake phobias, an important question that arises is whether prepared learning is tied to complete templates of the Jungian type or whether it stems from a genetically based fear of certain perceptual characteristics. Some observations suggest that the latter possibility might play a part. For example, Cornelius and Averill (1983) found that women frequently attribute their fear of spiders to the repulsive appearance and sudden movements of these animals. A study which examined this issue in more depth was carried out by Bennett-Levy and Marteau (1984). In this study, one group of subjects (n=62) were asked to indicate how afraid they were of 29 small animals. Another group (n=49) was invited to rate the same animals in terms of ugliness, sliminess, speediness, and suddenness of movement. A correlational approach was then carried out to explore whether the rated characteristics and self-reported fears covaried. Again, it was found that some animals (e.g., rat, spider, and snake) elicit more fear than others (e.g., ladybird, rabbit). In addition, the rank-order correlations strongly suggested that the more an animal is perceived as ugly, slimy, speedy or moving suddenly, the more that animal is feared. On the basis of these data, Bennett-Levy and Marteau argued that it is possibly perceptual characteristics of certain animals rather than these animals **per se** that humans are prepared to fear.

3.2 Snakes and chocolates: Clinical studies

There are clinical cases in which a preparedness interpretation has some **prima facie** plausibility. Other cases seem definitely "unprepared", no matter how much "imaginative reconstruction" is used. The case cited earlier (section 2.1) of the girl who developed a persistent phobia of snakes when her hand was caught in a closing car door (Marks, 1977) is a good example of the first category of phobic fears. The patient who acquired a phobia of chocolates when she saw a bar of chocolate in the room containing her mother's coffin (Rachman & Seligman, 1976) belongs to the second category. It is impossible to attach "survival value" to this peculiar fear.

The preparedness hypothesis does not claim that all phobias can be interpreted in terms of phylogenetic survival relevance (Rachman & Seligman, 1976). It only suggests that the great majority of phobias can be constructed in this way (Seligman, 1971). So far, three studies have been published which seem to sustain this claim (De Silva et al., 1977; De Silva, 1988; Zafiropoulou & McPherson, 1986). All of them employed a 5-point scale of preparedness (De Silva et al., 1977), with 1 indicating "unprepared" and 5 indicating "highly prepared", in order to enable independent judges to rate clinical cases for degree of preparedness.

Table 1. CLINICAL STUDIES CONCERNED WITH THE PREVALENCE OF PREPARED PHOBIAS.

Authors	Area	Sample Size	Type of Patients	Inter-Rater[2] Agreement	% Prepared[3]
De Silva et al.,1977	London	49	Phobias	0.78	96
Idem	Idem	82	Obsessions	0.92	84
Zafiropoulou & McPherson, 1986	Scotland	49	Phobias	0.95	69
De Silva, 1988	Sri Lanka	88	Phobias	0.79	90

[2] for preparedness ratings of fear content.

[3] % phobias which received a mean score of 2.5 or over.

De Silva et al. (1977) and Zafiropoulou and McPherson (1986) examined the records of an English and a Scottish sample of patients respectively (see Table 1 for details). Both studies found that a majority of the clinical fears were prepared, which means that they involved stimuli that were probably dangerous to pretechnological man. This finding was replicated by De Silva (1988) in a non-Western clinical population.

On the basis of Seligman's description of prepared learning (section 2.1), one would expect that prepared phobias are more quickly acquired, more severe, and more resistant to extinction than unprepared phobias (Rachman & Seligman, 1976; De Silva et al., 1977). In other words, strong correlations between preparedness ratings and clinical features such as mode of onset, treatment intensity, and treatment outcome would be predicted. Yet, none of the aforementioned clinical studies reported a significant relationship between the extent to which phobias are prepared and these clinical characteristics. Although the absence of a connection between clinical features and preparedness seems to contradict Seligman's description of prepared phobias, De Silva et al. (1977) remarked that this might be the result of a sampling bias. Seligman's hypothesis refers to the whole range of fear intensities, which vary from mild analog fears to severe clinical phobias. Therefore, one serious problem of the clinical studies is possibly that, because they usually concentrate on the severe cases in this range, differences in severity, treatment outcome, and mode of onset are too small to detect any associations with the degree of preparedness.

On the basis of their ethological analysis, Öhman et al. (1985) ascribed different characteristics to social and animal phobias (section 2.3). More specifically, they predicted that the age of onset would be earlier for animal than for social phobias. It was also predicted that animal phobias would be associated with more autonomic responding than social phobias. In a clinical study, Öhman et al. (1985) found some evidence to support these predictions. The onset ages of animal (N=40) and social (N=41) phobias were significantly different, the means being 7.3 and 15.5 years respectively. Additionally, animal phobics showed stronger cardiovascular responses than social phobics when they were confronted with their respective feared objects.

3.3 Illusory correlations and preparedness

Marks (1977) reported a case of a woman who happened to look at a photograph of a snake when the car in which she was travelling as a passenger became involved in an accident. Afterwards, this woman developed a phobia of snakes, not of cars. This anecdotal evidence suggests that humans tend to form selective associations when, by coincidence, "phylogenetically dangerous" stimuli and aversive experiences occur simultaneously. Using the illusory correlation paradigm, Mineka and Tomarken (1989) succeeded in establishing a laboratory model of these selective associations.

In the illusory correlation paradigm the subject is confronted with a series of trials, in which each trial consists of a stimulus (S) followed by an outcome (O). After the experiment, the subject is asked to estimate the conditional probability of each type of outcome given the prior occurrence of a certain stimulus [p(O/S)]. When the estimated probabilities diverge from the real probabilities, the subject is said to have formed a biased association.

In the first illusory correlation experiment that was carried out by Mineka and Tomarken (1989; Exp. 1), three types of stimuli were used; slides of snakes or spiders (phobia-relevant stimuli), slides of mushrooms, and slides of flowers. Each type of stimulus was followed by either an aversive outcome (electric shock), or a non-aversive outcome (tone), or by no outcome at all, for an equal number of times. As a result, conditional probabilities were .33 for all possible combinations of stimuli and outcomes. However, the normal subjects participating in this study (N=45) significantly overestimated the contingency between phobia-relevant stimuli and electric shock. This was true of both subjects low in snake or spider fear and subjects high in snake or spider fear, though the latter group displayed a particularly strong bias.

To examine whether this bias effect is attributable to the aversiveness or the salience of the electric shock, a second experiment was performed (Mineka & Tomarken, 1989; Exp. 2). In this experiment, the tone outcome was replaced by an attention-attracking but non-aversive compound stimulus consisting of a ringing chime and flashing lights. Although subjects rated this outcome and the shock outcome as being equally

salient, the conditional probability results indicated that high fear subjects (but not low fear subjects) still demonstrated a robust overestimation of the contingency between phobia-relevant stimuli and shock outcomes.

In a third experiment, Mineka and Tomarken (1989; Exp. 3) demonstrated that essentially the same results are obtained when there is an interval of a week between the experiment proper and the estimates by the subjects. They concluded that the overestimation of the contingency between phobia-relevant stimuli and aversive outcomes, particularly as present in high-fear subjects, may be

> "an important factor acting to promote the maintenance or exacerbation of fears and phobias over time" (p. 208).

3.4 Constructing a laboratory model: Conditioning studies

Aside from introducing the concept of preparedness, Seligman (1971) also suggested an experimental method for exploring this concept in more detail. He proposed to

> "do fear conditioning not with a tone or a light, as is usual, but with more natural CSs such as the picture and sound of snakes (...)" (p. 315/316).

Some 4 years later, Öhman and colleagues published their first reports of conditioning studies in which Seligman's proposal was realized (Öhman, Erixon & Löfberg, 1975; Öhman, Eriksson & Olofsson, 1975). In the two studies, normal subjects were confronted with either phobia-relevant CSs (i.e., slides of snakes) which were followed by an electric shock or neutral CSs (e.g., slides of houses) which were followed by a shock. The results indicated that skin conductance responses (SCRs) conditioned to phobia-relevant CSs extinguished more slowly than SCRs conditioned to neutral CSs. Since Seligman (1971) hypothesized that prepared learning is associated with delayed extinction, the first studies by Öhman were welcomed as strong support of the preparedness concept (De Silva et al., 1977). Indeed, these results were considered to be so encouraging that

Öhman and colleagues carried out a series of conditioning studies to test the predictions of the preparedness hypothesis systematically.

For appreciating the studies by Öhman and co-workers, a few words regarding the technical aspects of classical conditioning are in order. The typical aversive conditioning procedure consists of three phases. It usually starts with a habituation phase during which CSs are presented alone, that is, without subsequent UCS. This phase is necessary in order to eliminate preconditioning differences in responding to the CSs. A CS is regarded as a "prepotent" (Marks, 1987) or "innate fear" stimulus (Gray, 1982) if it elicits strong orienting responses during the habituation phase that do not readily decline. During the second phase, termed acquisition, the CSs are paired with an aversive UCS such as an electric shock several times. Occasionally, one-trial conditioning, which is a single CS-UCS pairing, is used. If the UCS follows immediately upon CS offset the conditioning technique is called a delay paradigm. A trace paradigm refers to a situation in which there is a lapse of time between CS offset and UCS onset (Prokasy & Kumpfer, 1973). The acquisition phase allows the comparison of acquisition rates of responses conditioned to different CSs. During the extinction phase the CSs are, again, presented several times without accompanying UCSs. The purpose of this phase is to determine to what extent conditioned responses disappear. Finally, it should be mentioned that there are two types of conditioning procedures (Prokasy & Kumpfer, 1973). In the single-cue procedure, the subject is confronted with only one CS. In the differential procedure the subject is exposed to two CSs, one of which (CS+) is followed by a UCS and the other one (CS-) is never followed by a UCS.

Usually following a differential conditioning procedure of the delayed type, Öhman and co-workers compared SCRs conditioned to slides of snakes and spiders (phobia-relevant CSs) to SCRs conditioned to neutral CSs in their first series of studies (see Table 2). By the last months of 1977, they had performed 8 experiments of this type. In 3 out of 8 studies, it was found that phobia-relevant CSs elicit greater SCRs during habituation than neutral CSs do. In only 2 out of 8 studies, it was reported that the phobia-relevant CSs affect acquisition rates to the extent that they are associated with faster response acquisition than neutral CSs. Yet, in 8 out of 8 studies a fear-relevance effect occurred

during extinction. That is, in all studies it was reported that during extinction, SCRs conditioned to phobia-relevant CSs disappear more slowly than SCRs conditioned to neutral CSs (see review by Öhman, Fredrikson & Hugdahl, 1978a). By 1985, the Öhman group had carried out over a dozen studies along the lines specified above (see also Table 3). They concluded that the fear-relevance effect during extinction "has been replicated over and over again" (Öhman et al., 1985; p. 38).

Öhman and associates usually obtained fear-relevance effects during extinction with differential (e.g., Öhman, Fredrikson, Hugdahl & Rimmö, 1976; Exp. 1), but occasionally also with single-cue (Öhman, Eriksson & Olofsson, 1975) conditioning paradigms. These fear-relevance effects were documented for delay (e.g., Öhman, Fredrikson & Hugdahl, 1978c) but also for trace (Hugdahl & Öhman, 1980) set-ups. Additionally, Öhman and associates reported that they are not limited to SCRs but can also be found when finger pulse volumes (FPV) (Fredrikson & Öhman, 1979; but see also Öhman, Eriksson & Olofsson, 1975) and electromyographic (EMG) activity at the corrugator region (Dimberg, 1987) are measured. The findings regarding heart rate (HR) are more equivocal: Whereas Fredrikson and Öhman (1979) failed to observe a conditioned cardioacceleration to phobia-relevant CSs, Fredrikson (1981)[4] reported some evidence suggesting that such a response might occur during acquisition. Recently, Cook, Hodes, and Lang (1986) reported that, during acquisition, accelerative responding is associated with phobia-relevant CSs but not with neutral CSs. However, none of the studies employing HR as a dependent variable succeeded in finding differential cardiac responding to phobia-relevant and neutral CSs during extinction. Finally, Öhman and colleagues claimed to have demonstrated that the fear-relevance effects during extinction do not depend on the salience of the phobia-relevant CS (Öhman et al., 1976; Exp. 2) and that they occur with phylogenetic fear stimuli (snakes and spiders) but not with ontogenetic fear stimuli (i.e., slides of broken electric equipment; Hugdahl & Kärker, 1981). All in all, the above-mentioned studies seemed to yield strong support for the assumption that prepared learning is characterized by slow extinction.

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[4]As this study strongly deviates from the usual conditioning paradigm it does not appear in Table 2.

Table 2. CONDITIONING STUDIES OF THE ÖHMAN GROUP IN WHICH SLIDES OF SNAKES OR SPIDERS (OCCASIONALLY ALSO SLIDES OF RATS) SERVED AS PHOBIA-RELEVANT CSs AND SLIDES OF MUSHROOMS OR FLOWERS (NOW AND THEN ALSO SLIDES OF HOUSES, CIRCLES, TRIANGLES, ELECTRIC OUTLETS, OR BERRIES) SERVED AS NEUTRAL CSs.

Study	Conditioning Paradigm	Response	Prepotency Effect Habituation	Fear-Relevance Effect Acquisition	Fear-Relevance Effect Extinction
Öhman, Eriksson & Olofsson, 1975	single cue\delay	SCR	+	-	+
		FPV	-	-	-
Öhman, Erixon & Löfberg, 1975	differential\delay, instructed extinction	SCR	?	-	+
Fredrikson et al., 1976	differential\delay	SCR	+	+	+
Öhman et al., 1976, Exp.1	differential\delay	SCR	-	-	+
Exp.2	differential\delay	SCR	-	-	+
Hugdahl & Öhman, 1977	differential\delay, instructed extinction	SCR	-	-	+
Hugdahl et al., 1977	differential\delay	SCR	+	+	+
Hugdahl, 1978	differential\delay, instructed extinction	SCR	?	-	+
Hygge & Öhman, 1978	differential\delay, vicarious acquisition	SCR	+	-	+
Öhman, Fredrikson & Hugdahl, 1978c	differential\delay	SCR	-	+	+

Table 2. Continued

Study	Conditioning Paradigm	Response	Prepotency Effect Habituation	Fear-Relevance Effect Acquisition	Fear-Relevance Effect Extinction
Fredrikson & Öhman, 1979	differential\delay	SCR	-	-	+
		FPV	-	-	+
		HR	+	-	-
Hugdahl & Öhman, 1980	differential\delay, differential\trace	SCR	-	-	+
Hugdahl & Kärker, 1981	differential\delay	SCR	-	-	+

Note. SCR: Magnitude or probability of the first-interval anticipatory component of the skin conductance response. FPV: finger pulse volume. HR: heart rate.

A plus sign (+) indicates that compared to neutral CSs, phobia-relevant CSs elicited greater responding during habituation (prepotency), or were associated with a faster acquisition rate of conditioned responses, or were associated with a delayed extinction of conditioned responses. A minus sign (-) indicates the absence of differences between CS+ and CS- linked responses. A question mark (?) indicates that relevant data were not published.

Other results obtained by the Öhman group are less clearly related to the preparedness hypothesis. For the sake of completeness, they are briefly summarized here. Öhman, Fredrikson and Hugdahl (1978c) reported that fear-relevance effects are found when SCRs are recorded at the palmar region of the hand but fail to occur when SCRs are recorded at the dorsal region (see also Fredrikson, 1981). Relating palmar activity and dorsal activity to defensive and orienting reactions respectively (Graham, 1973; Edelberg, 1973), Öhman and co-workers take their findings to mean that defensive responses rather than orienting responses are involved in aversive, prepared learning. As for the relationship between organismic factors and fear-relevance effects, Hugdahl et al. (1977) reported that these effects are particularly pronounced in highly aroused subjects (i.e., subjects with a large amount of spontaneous electrodermal fluctuations). In spite of the clearly documented sex differences in the incidence of animal phobias (Marks, 1969), Fredrikson et al. (1976) found that responses of males conditioned to snakes or spiders were as resistant to extinction as responses of females conditioned to these CSs.

In a number of studies, Öhman and co-workers examined whether, apart from delayed extinction, other characteristics of prepared learning (selectivity, quick acquisition, and non-cognitivity) could be demonstrated. The main findings of these studies can be summarized as follows:

1. The fear-relevance effect is not exclusively linked to slides of snakes and spiders; it can also occur when slides of angry faces are employed as CSs (e.g. Öhman & Dimberg, 1978; see Table 3). In the former case it is believed to tap selective learning that stems from the predatory defense system. In the latter case it is thought to be indicative of the social submissiveness system (Öhman, 1986; section 2.3).
2. While the Öhman group only found fear-relevance effects during acquisition (i.e., better or faster formation of conditioned responses to phobia-relevant than to neutral CSs) in a minority of their experiments, they also reported that a single pairing of a phobia-relevant CS with a UCS (i.e., one-trial conditioning) is sufficient to induce a subsequent resistance to extinction. A single pairing of a neutral CS with shock did not result in reliable conditioning effects, let alone in delayed extinction of conditioned responses.

Table 3. CONDITIONING STUDIES OF THE ÖHMAN GROUP IN WHICH SLIDES OF ANGRY FACES SERVED AS PHOBIA-RELEVANT CSs AND SLIDES OF HAPPY OR NEUTRAL FACES SERVED AS CONTROL CSs.

Study	Conditioning Paradigm	Response	Prepotency Effect Habituation	Fear-Relevance Effect Acquisition	Fear-Relevance Effect Extinction
Öhman & Dimberg 1978, Exp.1	differential\delay	SCR	-	-	+
Exp.2	differential\delay	SCR	-	-	+
Dimberg, 1987	differential\delay	SCR	-	-	+
		HR	-	-	+
		EMG	-	-	+

Note. EMG: electromyographic activity from the corrugator region.
See Table 2 for list.

The conditioning studies by Dimberg and Öhman (1983; Exp. 1, Exp. 2) and by Dimberg (1986a; Exp. 1 through Exp. 4) do not appear in this table because no comparisons between angry and happy facial stimuli were made (Dimberg & Öhman, 1983) or because stimulus conditions were not kept constant between acquisition and extinction (Dimberg, 1986a).

Accordingly, Öhman, Eriksson, and Olofsson (1975) concluded that fear-relevance effects during extinction do not require extensive Pavlovian acquisition. Moreover, Pavlovian conditioning (CS-UCS pairing) is not the only pathway along which fear-relevance effects can be induced. These effects also appear when "acquisition" merely consists of a model reacting fearfully to the phobia-relevant CS (Hygge & Öhman, 1978) or when it consists of verbal threats about a shock UCS (Hugdahl, 1978).

3. Fear-relevance effects emerge, even when "no more shock" instructions are given to the subject and shock-electrodes are removed prior to the extinction phase (Öhman, Erixon & Löfberg, 1975; Hugdahl, 1978; Hugdahl & Öhman, 1977; see Öhman & Hugdahl, 1979 for review). On the basis of this finding, Öhman and colleagues argued that fear-relevance effects are of a non-cognitive nature and, consequently, provide a laboratory analogue to the irrationality of phobic fears.

Referring to the above-mentioned results, Öhman and co-workers claimed to have developed a laboratory model for the prepared learning that is involved in animal and social phobias. For example, Öhman (1979) concludes

> "These data, then, provide an important link in the argument that autonomic responses aversively conditioned to potentially phobic stimuli provide a good model of phobic fear" (p. 123).

3.5 Animal studies

Various experimental psychologists of the second generation were highly familiar with the different types of fears displayed by monkeys. For example, Köhler (1925) showed that chimpanzees avoid social isolation, Yerkes and Yerkes (1936) gave a detailed description of snake fears in chimpanzees, Hebb and Riesen (1943) speculated about the origins of fear of strangers in chimpanzee infants, and Hebb (1946) drew attention to monkeys' fear of detached heads.

When reading these research papers, one is impressed by the subtlety of the arguments when it comes to explaining the etiology of chimpanzees' fears. Hebb (1946), for example, convincingly argued that an explanation

that is either based on "innateness" or on "conditioning" as the critical factor is doomed to fail. Nevertheless, in the case of chimpanzees' fear of snakes, he concluded that

> "It is best to let such fears stand for the present as not fully accounted for" (p. 272).

In a recent series of studies, Mineka and colleagues (Cook & Mineka, 1987; Mineka, 1987) sought to illuminate the etiology of snake fears in rhesus monkeys. Basically, most of their experiments consisted of a vicarious conditioning paradigm in which laboratory-reared monkeys ("observers") with no prior fear of snakes observed wild-reared monkeys ("models") reacting fearfully in the presence of a snake. After these vicarious conditioning trials, observers were usually found to have acquired an extremely persistent fear of snakes; even at a follow-up test session three months later, this fear showed no signs of diminution.

Mineka and co-workers presented evidence in favor of the view that observational conditioning of snake fears in monkeys is quite similar to direct classical conditioning. For example, Cook and Mineka (1987; Exp. 1) demonstrated that second-order conditioning may occur in vicariously acquired fears. Additionally, Mineka (1987) reported that the fears of observers correlated positively with the intensity of the fear displayed by the models. This seems to imply that the fear exhibited by the model acts as a UCS eliciting an unconditioned arousal response in the observer.

Using the vicarious conditioning paradigm, Mineka and colleagues carried out two experiments that are highly relevant to the issue under consideration here. In the first experiment (Cook & Mineka, 1987; Exp. 2), they demonstrated that when models react fearfully to a compound stimulus consisting of a snake and a flower, the observers' fear response on subsequent test trials is directed at the snake and not at the flower component. Obviously, the snake overshadows the flower stimulus. In the second, and most important, experiment (Mineka, 1987), observer monkeys (N=26) that were not afraid of snakes or spiders during a pretest, watched one of two videotapes. On the first tape, two model monkeys reacted fearfully to a toy snake but not to an artificial flower. With

the help of videosplicing techniques, another tape was constructed in which the same models reacted fearfully to the flower stimulus but did not do so to the toy snake. Thus, except for the direction of the fear, the two videotapes were matched on every variable (models, amount of fear displayed by the models, "animateness" of the stimuli, etc.). After the observer monkeys had watched one of the two videotapes during 12 sessions, a test session was held. The results of this session showed that observers watching the first version acquired a fear of snakes but not of flowers, whereas observers watching the second version did neither acquire a fear of snakes nor a fear of flowers. This learning bias can not be accounted for in terms of latent inhibition, since the monkeys had "no prior knowledge of or experience with either snakes of flowers" (Cook & Mineka, 1987; p. 363). Moreover, it was replicated in two follow-up studies (Mineka, 1988) and, consequently, seems to be a reliable phenomenon.

The results reported by Mineka are reminiscent of the study by Hygge and Öhman (1978) on preparedness and observational conditioning (section 3.4). Both studies suggest that the observational conditioning of fear is only effective when stimuli such as snakes are used. As for the theoretical interpretation of this selective, vicarious conditioning, Mineka (1988) agrees with Seligman (1971) in that she attributes it to a phylogenetically based readiness to associate fear with stimuli that may have posed a threat to early ancestors.

CHAPTER FOUR

REPLICATIONS AND CROSS-VALIDATIONS[1]

The previous chapter summarized clinical and experimental studies in favor of the preparedness hypothesis. Replications and cross-validations are, as Eysenck (1987b) stated, the life-blood of science. Therefore, a balanced evaluation of the empirical creditability of the preparedness hypothesis should take attempts to replicate and cross-validate the studies mentioned before into account. Chapter 6 will present an extensive review of these attempts. The scope of the present chapter is more limited. It contains some examples of studies that aimed at verifying the main results and assumptions of the earlier empirical work concerned with preparedness and phobias.

The following sections consist of separate research papers. Consequently, a certain amount of repetition and redundancy is inevitable. In passing, it should be noted that some sections, notably 4.1 and 4.3, were written at a stage at which data from other laboratories had not yet been published. Hence, a rather optimistic evaluation of the preparedness hypothesis dominates these sections now and then.

The first two sections deal with fear survey studies based on normal subjects (Merckelbach, van den Hout & van der Molen, 1987b; Merckelbach, van den Hout, Jansen & van der Molen, 1988). In the next two sections, more clinically oriented studies are presented (Merckelbach, van den Hout, Hoekstra & van Oppen, 1988; Merckelbach, van Hout, van den Hout & Mersch, 1989). Next, three psychophysiological conditioning experiments (Merckelbach, van der Molen & van den Hout, 1987; Merckelbach & van den Hout, 1988; Merckelbach & van den Hout, 1989) follow. The final section (4.8) summarizes the main findings of the empirical studies presented in this chapter.

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[1] Except for sections 4.7 and 4.8, all sections of this chapter have been published or accepted for publication.

4.1 Fear of animals: Correlations between fear-ratings and perceived characteristics

Summary.—Seligman (1971) proposed that, as a result of evolutionary processes, there is a genetic predisposition or preparedness to fear certain animals (e.g., snakes and spiders). Experimental and clinical studies have confirmed the notion of prepared fears of animals. Do prepared fears pertain to complete memory representations of these animals or are they related to specific, salient qualities that subjects attribute to these animals? A questionnaire designed to measure fear and avoidance of 30 small animals was administered to one group of 155 students while the characteristics of these animals (e.g., visual appearance, suddenness of movement, speediness) were rated by a second group of 116 students. For fear and avoidance as well as the perceived characteristics dimensions the animals were ranked on the basis of the scores that were given to them by the subjects. Spearman rank correlations of self-reported fear and avoidance with perceived characteristics were significant. This result suggests that subjects are prepared to associate fear not with complete representations of animals but with specific characteristics attributed to these animals.

According to Seligman's hypothesis of preparedness (1971), there is a genetically based or prepared tendency to associate fear and aversion with a certain set of animals. This hypothesis states that fear of such animals as snakes and spiders has been of great importance to the survival chances of the human species. Natural selection has favored genetic endowment to react fearfully to potentially harmful animals. Clinical as well as experimental studies support the hypothesis of preparedness. De Silva, Rachman, and Seligman (1977), and recently Zafiropoulou and McPherson (1986), reported that a large majority of phobic and obsessive-compulsive patients have a fear of "evolutionally significant" objects or situations. Using electric shock as an aversive unconditioned stimulus and slides of snakes and spiders ("evolutionally significant") or of mushrooms and flowers ("evolutionally neutral") as conditioned stimuli, Öhman and colleagues [see reviews by Öhman, Fredrikson, and Hugdahl (1978) and Öhman (1986)] showed in several studies that the conditioned skin-conductance response of normals extinguishes slower with evolutionally significant than with evolutionally neutral stimuli.

Supporters of the hypothesis of preparedness (Seligman, 1971; Eysenck, 1979; Mineka, 1985) have not specified the mechanism on which prepared fears are based. From their accounts one could easily get the impression that evolutionary processes have provided humans with complete, built-in memory representations (phobia prototypes; see Lang, 1984) of, say, snakes and spiders which readily acquire a negative emotional value. Studies of fear in animals (Schneirla, 1965; Hinde, 1974), however, demonstrated that some, presumably innate, fear reactions are elicited by specific stimulus configurations, e.g., speed, suddenness of movement, strange appearance. On the basis of these studies, Bennett-Levy and Marteau (1984) reasoned that, in man, prepared fears may also be related to certain stimulus configurations rather than to complete representations. In their study, one group of subjects rated how fearful they were of various animals while another group rated the characteristics of these animals on various perceptual dimensions. High correlations were found between ratings of fear and perceived characteristics. The authors claimed to have shown that "ugly, slimy, speedy, or sudden-moving animals are experienced as less approachable and more fear-provoking than animals without these qualities" (p. 40). This finding is in line with a reformulation of the hypothesis of prepared fears in terms of perceived characteristics.

However, several questions emerge. The first has to do with the fact that Bennett-Levy and Marteau instructed their second group of subjects "to consider how ugly, slimy, and speedy the animals are and how suddenly they appear to move." So the dimensions used pertained only to the visual and tactile modalities. Are olfactory and auditory impressions also associated with self-reported fear? A second, more important question concerns the negative connotations especially of "sliminess" and "ugliness." Is it possible that the correlations reported by Bennett-Levy and Marteau simply are a reflection of this negative emotional tone? It may also be reasoned that sliminess and ugliness represent degrees of "strangeness." In that case, the observed relation of sliminess and ugliness with fear reflects xenophobic predispositions of the kind documented by Hebb (1946). Conceptually related to earlier work on xenophobia are more recent studies showing that, in general, organisms prefer predictable and controllable events to unpredictable and uncontrollable ones (Seligman, Maier, & Solomon, 1971; Mineka & Kihlstrom, (1978). In the evolutionary context of the hypothesis of prepared fears xenophobic predispositions and a preference for predictability and controllability make good sense.

With all of these arguments in mind, we repeated the Bennett-Levy and Marteau study. The dimensions to be scored, however, concerned all four perceptual modalities and, in merely asking subjects to rate strangeness in these modalities, there were no or at least fewer *a priori* negative connotations. In addition, we asked our subjects to indicate how predictable and controllable they thought the various animals were.

METHOD

Subjects

Students taking courses in the health sciences at the University of Limburg volunteered to complete either a fear questionnaire or a questionnaire asking for perceived characteristics and controllability/predictability. All questionnaires were randomly distributed.

The 155 (34 men and 121 women) students who completed the fear questionnaire constituted Group 1. Their mean age was 23.4 yr. Group 2 consisted of the 36 men and 80 women who completed the questionnaire on perceived characteristics and controllability/predictability. Their mean age was 22.1 yr. The higher proportion of women in both groups is a consequence of the skewed sex distribution of the student population taking courses in the health sciences at this university.

Materials

The fear questionnaire measured self-reported fear and avoidance of 30 small animals. The items, shown in Table 1, were derived from the Bennett-Levy and Marteau study. As in that study the written instructions stressed that all animals had to be regarded as "harmless" representatives of the various animal species. To enhance compatibility with their study, we used their rating scales: fear of each animal was rated on a 3-point scale (1 = not afraid, 2 = somewhat afraid, 3 = very afraid). In addition, avoidance was rated on a 5-point scale (1 = would enjoy picking it up, 2 = would pick up but unpleasant, 3 = would touch it or go within six inches of it, 4 = would stand one to six feet away from it, 5 = would stand further than six feet away from it).

The perceived characteristics questionnaire was constructed to measure the impressions that the same 30 animals made on the students in Group 2. The introduction to this questionnaire was as follows: "We would like you to judge to what extent the following animals look strange, feel odd when you touch them, make strange noises and have a peculiar smell." Further, we asked the subjects to what extent they believed that the animals listed move suddenly, are speedy, and are controllable and predictable in their behavior. The subjects gave all their judgements on a 3-point scale (1 = not at all, 2 = somewhat, 3 = very).

RESULTS

An analysis of variance approach was used to estimate the reliability of the dimensions (Winer, 1962). Reliabilities in terms of Cronbach's *alpha* for the dimensions fear, avoidance, visual appearance, tactile impression, auditory impression, odor, suddenness of movement, speediness, controllability, and predictability were, respectively, .90, .92, .92, .94, .86, .94, .90, .87, .94, and .95.

TABLE 1

MEAN RATINGS OF GROUP 1 (FEAR AND AVOIDANCE) AND GROUP 2 (VISUAL APPEARANCE, TACTILE IMPRESSION, AUDITORY IMPRESSION, ODOR, SUDDENNESS OF MOVEMENT, SPEED, CONTROLLABILITY, AND PREDICTABILITY) FOR 30 SMALL ANIMALS

Animal	Group 1		Group 2							
	1*	2	3	4	5	6	7	8	9	10
grass snake	2.4	4.3	1.9	2.4	1.7	1.3	2.3	2.3	1.4	1.4
rat	2.2	4.1	1.6	1.8	1.4	1.8	2.3	2.4	1.6	1.6
jellyfish	1.9	4.0	2.5	2.8	1.1	1.4	1.3	1.2	1.8	1.8
cockroach	1.8	3.7	2.2	2.3	1.5	1.4	1.8	1.7	1.8	1.7
spider	1.8	3.4	2.0	2.2	1.1	1.1	2.0	1.9	1.8	1.7
lizard	1.6	3.1	1.9	2.1	1.4	1.3	2.3	2.3	1.5	1.5
bee	1.6	3.7	1.4	1.9	1.5	1.1	2.1	2.1	1.6	1.7
mouse	1.4	2.9	1.2	1.4	1.3	1.4	2.5	2.6	1.7	1.7
grasshopper	1.4	2.9	1.9	1.9	1.7	1.1	2.6	2.3	1.6	1.5
crow	1.4	2.8	1.2	1.4	1.6	1.2	2.0	2.1	1.7	1.7
beetle	1.4	2.5	1.8	2.0	1.4	1.3	1.7	1.6	2.0	1.8
chimpanzee	1.3	2.3	1.2	1.3	1.6	1.7	1.8	2.0	1.9	2.0
frog	1.3	2.8	1.9	2.4	1.7	1.4	2.4	2.0	1.6	1.6
moth	1.3	2.6	1.6	1.8	1.2	1.3	1.8	1.7	1.7	1.7
worm	1.2	2.5	1.8	2.3	1.1	1.2	1.2	1.1	2.0	1.8
caterpillar	1.2	2.3	1.9	2.2	1.1	1.2	1.1	1.0	2.2	2.0
slug	1.2	2.3	2.0	2.4	1.1	1.3	1.1	1.0	2.2	2.1
cat	1.1	1.3	1.0	1.0	1.2	1.2	2.0	2.3	2.2	2.0
baby seal	1.1	2.1	1.3	1.6	1.6	1.4	1.3	1.5	2.0	1.9
spaniel	1.1	1.4	1.1	1.1	1.2	1.4	1.7	2.1	2.5	2.3
hamster	1.1	1.4	1.1	1.2	1.3	1.5	1.7	1.8	2.1	1.9
blackbird	1.1	1.8	1.1	1.3	1.2	1.1	1.9	2.0	1.7	1.8
ant	1.1	2.0	1.5	1.6	1.1	1.1	1.8	2.0	1.9	1.9
tortoise	1.1	1.7	1.5	1.6	1.1	1.3	1.0	1.0	2.3	2.1
robin	1.1	1.6	1.0	1.2	1.1	1.1	2.0	2.1	1.7	1.8
butterfly	1.0	1.5	1.2	1.5	1.1	1.0	1.9	1.8	1.8	1.7
squirrel	1.0	1.6	1.1	1.2	1.1	1.2	2.5	2.6	1.6	1.7
rabbit	1.0	1.2	1.0	1.1	1.1	1.3	2.2	2.4	2.0	2.0
lamb	1.0	1.3	1.0	1.1	1.3	1.3	1.6	1.7	2.2	2.0
ladybird	1.0	1.1	1.3	1.3	1.0	1.1	1.7	1.5	2.1	1.8

Note.—Standard deviations ranged from 0.01 to 0.12, with most falling between 0.04 and 0.07. Detailed values are on file in Document NAPS-04511. Remit $8.65 for photocopy or $4.00 for fiche to Microfiche Publications, POB 3513, Grand Central Station, New York, NY 10017.
*See Table 2 for list.

Next, the scores of the subjects were averaged for each item and for each dimension. Thus, 30 × 10 mean scores were obtained. Table 1 gives the mean scores and standard deviations of Group 1 (fear and avoidance) and Group 2 (visual appearance, tactile impression, auditory impression, odor, suddenness of movement, speediness, controllability, and predictability) on

the animal items. As can be seen, subjects in Group 1 reported their highest fear and avoidance on the snake and rat items.

From the data of Table 1, Spearman rank correlations between the dimensions were computed (Siegel, 1956). Rank orders of the items were established for each dimension on the basis of the mean scores and the associations between these rank orders were computed (Table 2). As can be seen in Table 2, self-reported fear correlated significantly and positively with all perceived characteristics, except suddenness of movement and speediness. Self-reported fear correlated significantly and negatively with controllability and predictability. Ratings of avoidance too, correlated significantly with most perceived characteristics and scores on the controllability and predictability dimensions. Exceptions here were the correlations with odor and speediness.

TABLE 2

SPEARMAN RANK CORRELATIONS BETWEEN MEASURES OF FEAR AND AVOIDANCE AND PERCEIVED CHARACTERISTICS

	1*	2	3	4	5	6	7	8	9
2	.94†								
3	.69†	.75†							
4	.67†	.78†	.94†						
5	.55†	.48†	.14	.15					
6	.36†	.22	.16	.09	.45†				
7	.29	.31†	−.09	−.08	.46†	−.04			
8	.16	.11	−.37	−.37†	.39†	.02	.88†		
9	−.52†	−.64†	−.22	−.32†	−.50†	.08	−.80†	−.61†	
10	−.55†	−.66†	−.40†	−.43†	−.44†	.02	−.73†	−.44†	.89†

*1: fear, 2: avoidance, 3: visual appearance, 4: tactile impression, 5: auditory impression, 6: odor, 7: suddenness of movement, 8: speediness, 9: controllability, 10: predictability.
†$p \leq .0.05$, one-tailed, for $r \leq .31$, $n = 30$.

When controlling for appearance, the partial correlations of fear with suddenness of movement and speediness became significant ($r = 0.53$, $p < 0.001$ and $r = 0.62$, $p < 0.001$, one-tailed). The same held for the partial correlations of avoidance with smell and speediness when the influence of visual appearance was partialed out ($r = 0.34$, $p < 0.05$ and $r = 0.61$, $p < 0.001$, one-tailed).

Separate correlational analyses were carried out for men and women. The correlational pattern was, by and large, the same in both groups which did not differ significantly from that of the combined sample. However, two exceptions have to be noted. First, while the correlation between fear and odor remained low and nonsignificant ($r = 0.05$) for men, this correlation was significant for women ($r = 0.42$, $p < 0.01$, one-tailed). Second, for women, predictability was significantly related to fear ($r = 0.58$, $p < 0.001$, one-tailed), but for men this association was nonsignificant ($r = 0.29$).

Discussion

The data presented here show significant correlations of self-reported fear and avoidance of animals with perceived characteristics: the more an animal is experienced as "strange" on a particular dimension, the more that animal is feared and avoided. Although we gave rather neutral instructions to our subjects, the results confirm the correlation of fear and avoidance with tactile and visual characteristics that was reported by Bennett-Levy and Marteau (1984). Moreover, we investigated odor, auditory impression, predictability, and controllability as separate dimensions and observed that these dimensions too were significantly related to ratings of fear and avoidance. We found significant correlations of predictability and odor with fear for women but not men. We do not see plausible theoretical explanations for this and are reluctant to speculate about possible causes.

The high correlations between measures of fear and characteristics scores can be taken as support for a reformulation of the hypothesis of preparedness in terms of perceived characteristics. Subjects may be prepared to associate fear with certain salient qualities rather than with extensive representations of objects formerly relevant to survival. Admittedly, a radical distinction between objects *per se* and their stimulus characteristics is not meaningful since objects are ultimately composed of stimulus characteristics. Nevertheless, our results and those of Bennett-Levy and Marteau strongly suggest that fear of animals like snakes and spiders is focused on a limited set of characteristics of these animals. As Bennett-Levy and Marteau point out, this may have implications for the behavioral treatment of animal probias. It is conceivable that a desensitization procedure is more successful when it is directed at salient details, e.g., slimy skin, strange noise, etc., of the animal rather than the complete animal.

Experimental support for the hypothesis of preparedness stems mainly from the observation of Öhman and coworkers (1978, 1986) that the conditioned skin conductance response to slides of snakes and spiders is more resistant to extinction than the conditioned skin conductance response to slides of flowers and mushrooms. However, snakes and spiders differ from flowers and mushrooms in many more respects than just in the amount of survival relevance. From a perceived characteristics point of view, it would seem that the reported difference in conditioned skin conductance response extinction results from perceptual differences between the stimulus classes used. It would also follow that a difference in conditioned skin conductance response extinction will not occur if in Öhman-like set-ups the objects depicted by evolutionally significant and neutral slides have crucial qualities, such as strange appearance, suddenness of movement, unpredictability, etc., in common. Empirical dissection of the characteristics that typically define phobogenic stimuli will enable us to evaluate the merits of the hypothesis of preparedness.

It should be stressed that the present study was correlational in nature and that the functional direction of the observed association remains a matter for further investigation. It could be argued that the more an animal is feared, the more extreme will be ratings of the characteristics of this animal by the subject. Therefore, the question remains: Do specific characteristics provoke fear or does fear influence the perception of characteristics?

Supplying subjects with pictures of imaginary animals described as either dangerous or harmless and asking them to rate the animals' characteristics might provide an answer.

4.2 Many stimuli are frightening, but some are more frightening than others: The contributions of preparedness, dangerousness, and unpredictability to making a stimulus fearful

The nonrandom distribution of situational fears has been explained by evolutionary survival relevance of specific fears. Thirty-eight stimuli were taken from the literature on "preparedness" and were scored on fearfulness, objective dangerousness, and spatiotemporal unpredictability by three separate groups of students. The same items were scored on survival relevance by 15 biologists. Fearfulness of cues significantly correlated not only with survival relevance but also, and even more strongly, with dangerousness and unpredictability. While the fear/survival relevance association virtually disappeared when the "unpredictability" contribution was partialed out, the fear/unpredictability correlation was only marginally affected when controlling for survival relevance. This suggests that nonrandomness of feared stimuli may result from the spatiotemporal unpredictability that is attributed to these stimuli. The current practice of using snakes and spiders as "phobia-relevant," and flowers and mushrooms as "neutral," cues was not justified by the ratings of the 15 independent experts.

KEY WORDS: phobias; conditioning; preparedness.

INTRODUCTION

It is a well-established fact that some stimuli (e.g., snakes, thunder) more often become and remain frightening than do other stimuli (Agras, Sylvester, & Oliveau, 1969; Costello, 1982). This selectivity has been explained in terms of Darwinian theory: that is, fear of stimuli that were survival-relevant to our ancestors may have become genetically coded in the form of a predisposition or preparedness to associate readily fear with particular stimuli and may still give rise to phobias (Seligman, 1971).

Experimental support for the preparedness hypothesis comes mainly from the research of Öhman and colleagues. In a series of conditioning experiments, they paired an electric shock with slides of either phobia-relevant stimuli (usually spiders and snakes but recently also angry faces) or neutral stimuli (usually flowers and mushrooms but also happy faces). The extinction of the conditioned electrodermal responses to phobia-relevant slides was strongly delayed compared to the extinction of responses to neutral stimuli (see reviews by Dimberg, 1986; Öhman, 1986; Öhman, Dimberg, & Öst, 1985; Öhman, Fredrikson, & Hughdahl, 1978).

In the majority of their experiments, Öhman and co-workers used a differential conditioning paradigm. Studies in which a different conditioning procedure was followed, however, yielded no preparedness phenomena (Deitz, 1982; Emerson & Lucas, 1981; Vaitl, Gruppe, & Kimmel, 1985; Merckelbach & van den Hout, 1988; McNally, 1981, 1986; McNally & Reiss, 1982, 1984).

Moreover, efforts in other centers to replicate the Öhman *et al.* results have either failed (McNally & Foa, 1986; Merckelbach, van der Molen, & van den Hout, 1987) or only justified the conclusion that the preparedness phenomenon appears to be a "fragile" effect (Cook, Hodes, & Lang, 1986) or "borderline interaction" Dawson, Schell, & Tweddle Banis, 1986).

Apart from the phenomena reported by Ohman *et al.* needing cross validation from other centers and from other experimental paradigms, the preparedness hypothesis and, in its wake, the preparedness research present theoretical problems (see reviews by Merckelbach, van den Hout, & van der Molen, 1988; McNally, 1987). The preparedness hypothesis was formulated to account for the restricted range and nonrandom distribution of situational fears. This nonrandomness was supposed to be an inexplicable anomaly to Pavlovian conditioning theory, whose founder claimed that "any natural phenomenon chosen at will, may be converted into a conditional stimulus . . . any visual stimulus, any desired sound, any odor and the stimulation of any part of the skin" (Pavlov, 1928, p. 86). This view has been taken to imply that *all* stimuli are equally likely to become phobic cues. Admittedly, classical Pavlovian theory is heavily biased toward the conditioning of responses

to neutral conditioned stimuli (CS). However, as Gray (1979) rightly remarked, "it is worth asking what conditioning theory is *for*. Pavlov felt impelled to cross the great divide between the unconditioned and the conditioned reflex when he observed in the phrase cited by Eysenck (see also above) that 'any natural phenomenon chosen at will may be converted into a conditioned stimulus.' Had the salivary reflex been elicited only by tastes or smells closely related to nutritious substances, *there would have been no need to take this step*" (p. 70, our italics). It should not be overlooked that, while the theory does say that, *in principle,* any previously neutral stimuli may after pairing with an unconditioned stimulus (UCS), become a CS for anxiety, it also implies that whether *in fact* a stimulus often becomes a phobic cue depends on the degree to which the cue signals aversive events. Therefore, in order to be able to determine whether the preparedness hypothesis really has more to offer in terms of an explanation that "traditional" Pavlovian notions, it is necessary to examine how well the nonrandomness of situational fears can be attributed either to phylogenetic survival relevance or to ontogenetic dangerousness in the sense of signalization of objective danger.

Other problems which are addressed in the present study stem primarily from the experimental elaboration of the preparedness hypothesis. It was reported that classically conditioned electrodermal responses to slides of spiders and snakes extinguish more slowly than do such responses to slides of flowers or mushrooms. Before concluding that slower extinction to specific stimuli is due to survival relevance, it needs to be independently established that the two groups of stimuli do, indeed, differ considerably in this respect. Judgments on our evolutionary history are, by their very nature, post hoc and great care should be taken not to confound hypothetical evolutionary scenarios with researchers' bias: thus, our classification of stimuli as "phobia-" or "survival-relevant" should not be deduced from data on the prevalence of fears. To avoid begging the question, the labeling of some stimuli as survival-relevant should preferably not be done by anxiety researchers but rather by independent experts who may be less prone to a biased judgment.

Furthermore, it should be noted that the most frequently used stimulus cues in the psychophysiological research on preparedness – namely, spiders and snakes, on the one hand, and mushrooms and flowers, on the other hand – differ in many more respects than just in their degree of survival relevance. For one thing, the spatiotemporal unpredictability associated with snakes/spiders appears to be much higher than that of flowers/mushrooms. This may be of interest, as there are strong theoretical and experimental indications that unpredictable (poor probabilistic CS/UCS relation) or underpredicted aversive events can produce strong motivational and emotional effects (Arntz & van den Hout, 1987; Mineka & Kihlstrom, 1978; Rachman & Lopatka, 1986; Seligman, 1975). Unpredictable objects are relatively like-

ly to become and remain fear cues. Thus, spatiotemporal unpredictability might provide an alternative explanation for the nonrandomness of fears and of the Öhman *et al.* findings.

In sum, the present study investigates how closely the nonrandomness of fears is associated with (and might thus possibly be explained by) (a) the potential dangerous of the pertinent stimuli (i.e., the degree to which the stimulus signalizes objective harm), (b) the survival relevance of the fears for our prehistoric ancestors, and (c) the spatiotemporal unpredictability of stimuli.

METHOD

Questionnaires

A list of stimuli was constructed that would be rated on fearfulness, dangerousness, survival relevance, and unpredictability by four separate groups of subjects (see below). In the present context, the most appropriate selection of items was thought to include those stimuli that appear in the current literature on preparedness. Most of these items are often referred to by researchers as prototypical examples of stimuli that are prepared, unprepared, or even contraprepared to become associated with fear or aversion.

The following 38 items were selected: rabbit (Eifert & Schermelleh, 1985), pair of opera glasses (Valentine, 1930), rat, pajamas (Seligman, 1971), Béarnaise sauce, Wagner opera (Seligman & Hager, 1972), curtain (Bregman, 1934; cited by Seligman, 1971), horse (Freud, 1909; cited by Seligman, 1971), spider, snake, flower, mushroom (Öhman, 1986), electrical wire, electrical outlet (Hugdahl & Kärker, 1981), splintered glass, fatal number, wasp, social gathering, predator such as tiger,[3] unfamiliar place, darkness, blood, fire, animal feces (De Silva, Rachman, & Seligman, 1972), chocolate (Rachman & Seligman, 1976), household objects[3] such as coffeemill and scissors, handgun (Cook *et al.*, 1986), angry face, happy face (Dimberg, 1986), dirt (Turner & Michelson, 1984), enclosed place (Eysenck, 1979), dentist (Agras *et al.*, 1969), fearful face (Örr & Lanzetta, 1980), thunderstorm (Freud, 1926; cited by Rachman, 1978), lamb (Rachman, 1978), car door (Marks, 1977), and war-related stimulus such as siren[3] (Rachman, 1977; Saigh, 1984).

Scores for each of the four dimensions were given on a 10-cm visual analogue scale ranging from "not fearful" to "extremely fearful" (fearfulness dimension), "extremely unlikely" to "extremely likely" that the stimulus signals objective harm (dangerousness dimension), "highly predictable" to "not at all predictable" (unpredictability dimension), and "not survival-

[3]Sometimes stimulus classes are so abstractly described that they are inappropriate as items. In these instances, we used a concrete example of the stimulus class in question.

relevant at all" to "extremely survival-relevant" to prehistoric man (survival relevance dimension). The fearfulness dimension was constructed to measure self-reported fear of the 38 items. In the unpredictability and dangerousness versions, subjects were instructed to rate the unpredictability and dangerousness of the stimuli for people in general. As for the survival relevance dimension, the instructions given to the subjects were quite similar to those used by De Silva *et al.* (1972): subjects were asked to indicate to what extent fear of the stimuli listed facilitated survival of *Homo sapiens* in pretechnological society. Dimensions were scored by different groups of subjects in order to avoid that scoring on one dimension would influence scores on the other dimensions (i.e., experimentwise errors).

Subjects

The fear dimension, dangerousness dimension, and unpredictability dimension were scored by undergraduate students (fear dimension—N = 37, 7 men and 30 women, mean age = 20.6 years; dangerous dimension—N = 18, 2 men and 16 women, mean age = 21.1 years; unpredictability dimension—N = 14, 5 men and 9 women, mean age = 22.4 years). The survival relevance dimension was given to 15 human biology researchers (10 men and 5 women, mean age = 29.2 years) who were unaware of the subject under investigation and who were professionally initmately acquainted with Darwinian theory.

RESULTS

Internal Consistency of Questionnaires

An analysis of variance approach was used to estimate the reliability of the dimensions (Winer, 1962). It was decided beforehand that individual items which might contribute to a low internal consistency, as indicated by Cronbach's alpha for the total dimension of less than .70, would be excluded from further analysis. However, when all items were included, alpha coefficients for the four dimensions exceeded this criterion. Alpha coefficients for fearfulness, survival relevance, dangerousness, and unpredictability were .94, .95, .91, and .90, respectively.

Choice of Stimuli in Preparedness Research

As for the choice of stimuli in preparedness research, the following items are the most interesting ones because of their prominent place in prepared-

ness research: flower, spider, mushroom, snake, angry face, and happy face. Recalling that flowers and mushrooms are used as prototypes of neutral cues, while spiders and snakes are used as paradigmatic instances of phobia-relevant stimuli, one would expect that independent scores on survival relevance of snakes and spiders would be significantly higher than survival relevance scores on mushrooms and flowers.

While scores for snakes on survival relevance were, indeed, significantly higher than for flowers, the spider vs. flower difference was statistically not significant (see Table I). The mushroom vs. snake difference reached borderline significance. Interestingly, the independent judges rated fear of mushrooms as considerably more survival relevant than fear of spiders. This is in sharp contrast to current assumptions underlying experimental work in the realm of preparedness.

Fear of angry faces was rated as significantly more survival-relevant than fear of happy faces [$t(14) = 3.33$, $p < .05$, one tailed]. In the experimental research on preparedness, these stimuli have never been used in combination with mushrooms, flowers, snakes, or spiders. Therefore, no other comparisons were made. In most psychophysiological studies, responses to groups of phobia-relevant and supposedly neutral stimuli rather than responses to individual stimuli are compared to each other. Therefore, it was decided to cluster the items according to their actual use of phobia-relevant or neutral stimuli. Scores were averaged over the three most commonly used phobia-relevant items, namely, snake, spider, and angry face, and compared to mean scores of a group of neutral items (mushroom, flower, and happy face) on the dimensions fearfulness, survival relevance, dangerousness, and unpredictability (see Fig. 1). As expected, t tests revealed that the two groups of stimuli differed significantly on the fear dimension [$t(36) = 8.8$, $p < .05$,

Table I. Expert Judgment (0–100) of Survival Relevance of Four Widely Used Stimuli in preparedness Research: Means (M), Standard Deviations, and t Values for Relevant Comparisons

	Snake	Spider
	$M = 81.7$ SD = 20.9	$M = 45.3$ SD = 32.8
Mushroom $M = 69.1$ SD = 35.1	$t = 1.74^{*}$	$t = -2.76^{**}$
Flower $M = 34.4$ SD = 34.8	$t = 4.73^{**}$	$t = 1.05$

$^{*}p < .10$ one tailed, df = 14.
$^{**}p < .05$, one tailed, df = 14.

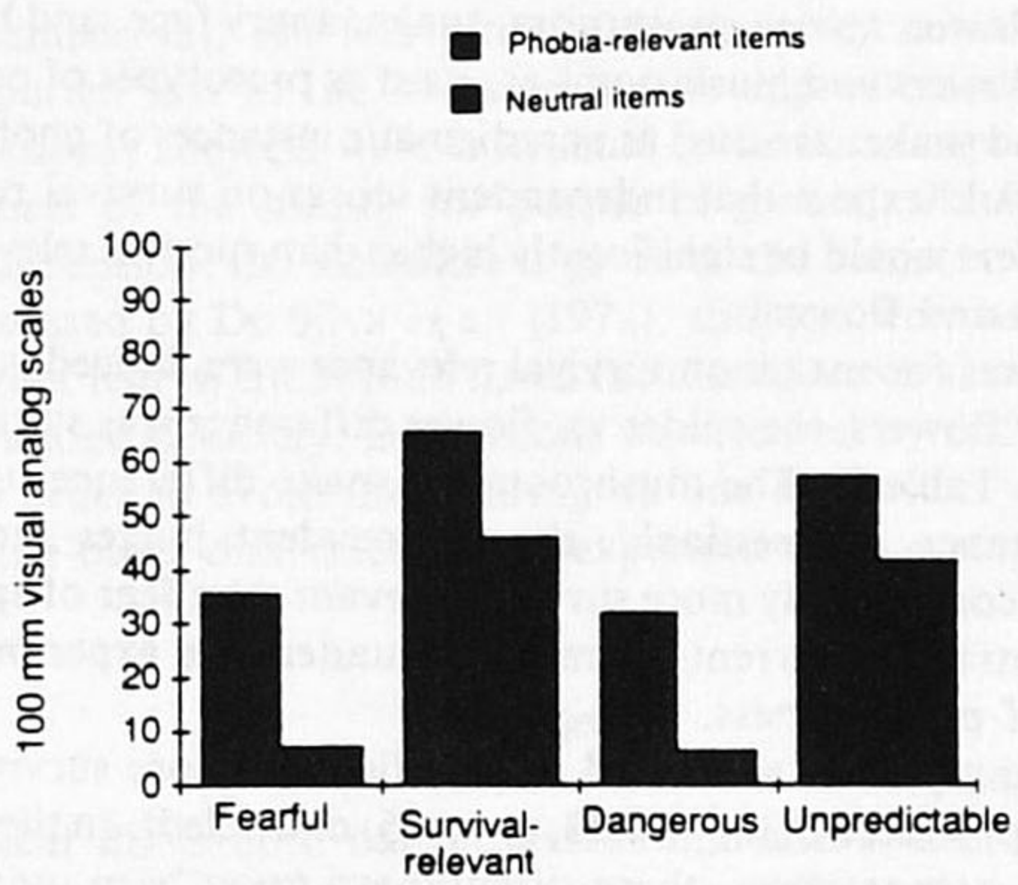


Fig. 1. Mean scores (0–100) combined for three widely used phobia-relevant items (snake, spider, angry face) and three widely used neutral items (mushroom, flower, happy face) on the dimensions fearfulness, survival relevance, dangerousness, and unpredictability.

one tailed]. Furthermore, a significant difference was found on the survival relevance dimension [$t(14) = 2.8, p < .05$, one tailed]. However, differences were also present, and even more markedly, on the unpredictability [$t(13) = 4.1, p < .05$, one tailed] and dangerousness [$t(17) = 5.4, p < .05$, one tailed] dimensions.

Nonrandomness of Fear: Associations with Danger Signal Value, Survival Relevance, and Spatiotemporal Unpredictability[4]

If nonrandomness and selectivity of fearful stimuli are due to survival relevance of the pertinent stimulus, one would expect a strong positive correlation between fear and survival relevance. Similarly, to the degree that objective dangerousness or unpredictability is responsible for nonrandomness, close associations should be expected between fear and dangerousness and/or fear and unpredictability. On the basis of the mean score on the 38 items, Pearson product-moment correlations among the four dimensions were computed. Results are shown in Table II.

[4]A table containing mean scores and standard deviations of all items on the four dimensions is available from the first author.

Table II. Pearson Product-Moment Correlations Between Averaged Ratings of 38 Items on Fearfulness, Survival Relevance, Dangerousness, and Unpredictability*

Dimension	Survival relevance	Dangerousness	Unpredictability
Fearfulness	.51	.87	.63
Survival relevance	–	.43	.64
Dangerousness	–	–	.51

*$p < .01$, one tailed, $N = 38$, for all reported correlations.

Fear correlated significantly with all three other variables. To determine how much unique variance can be accounted for by survival relevance, dangerousness, and unpredictability, an analysis with partial correlations was carried out. When the fear/unpredictability correlation was corrected for the contribution of survival relevance, the coefficient dropped to .47 but was still significant (df = 35, $p < .05$, one tailed). The fear/dangerousness correlation was only marginally affected when variance due to survival relevance was partialed out ($r = .85$ instead of .87, df = 35, $p < .05$, one tailed). However, a very different picture emerged when the fear/survival relevance association was corrected for dangerousness and unpredictability. Correcting for dangerousness reduced the fear/survival relevance coefficient from .51 to .31 (df = 35, $p < .05$, one tailed), while correcting for unpredictability reduced the fear/survival relevance association to the nonsignificant coefficient of .18.

Thus, the significant association between fear and unpredictability can be explained only partly by survival relevance, while survival relevance does not explain at all the high correlation between fear and dangerousness. On the other hand, the fear/survival relevance correlation drops to a nonsignificant level of .18 once the contribution of unpredictability of stimuli is taken into account.

A multiple regression analysis confirmed the above-mentioned findings. The proportion of variance (R^2) in fear explained by unpredictability, dangerousness, and survival relevance operating jointly was .82 [$F(3,34) = 50.0$, $p < .05$]. This proportion was hardly affected when only unpredictability and dangerousness were used as predictors: $R^2 = .81$ [$F(2,35) = 76.3$, $p < .05$]. The t values associated with the regression coefficients of unpredictability, dangerousness, and survival relevance were 8.6 ($p < .05$, two tailed), 2.1 ($p < .05$, two tailed), and .56 ($p = .57$), respectively.

DISCUSSION

In line with the Pavlovian view, fear of stimuli proved to be highly correlated with the degree to which these stimuli signalize objective harm. Fear

ratings were further correlated with unpredictability, while a somewhat smaller correlation was found between fear and survival relevance. This fear/survival relevance association virtually disappeared once the variance attributable to unpredictability was eliminated.

The expert judgments on survival relevance strongly diverged from current views and research practices. Mushrooms, for example, were judged to be highly survival-relevant. This result is compatible with the provocative view put forward by Delprato (1980). He stated that mushrooms "have posed a greater threat to the survival of the human species than have spiders and snakes combined. Mushroom toxicosis is especially a threat to humans because poisonous mushrooms are usually extremely difficult to discriminate from non-poisonous varieties" (p. 89).

It was found that, as a group, the most widely used phobia-relevant stimuli differ from neutral stimuli more in terms of dangerousness and unpredictability than in terms of survival relevance. At the same time, the overall correlational analysis revealed that the correlations of fear with dangerousness and unpredictability are relatively independent of the survival relevance dimension.

De Silva *et al.* (1977) stated that the nonrandom distribution of fear "forms the core of the preparedness concept" (p. 76). The studies by Agras *et al.* (1969) and by Costello (1982) are frequently cited to document the higher prevalence of biologically relevant fears than fears with an ontogenetic background. Yet as Kirkpatrick (1984) rightly remarks the exact rank order of fears that one observes in a population is highly dependent upon the choice of items that are included in a fear survey. Kirkpatrick found that fear of death of a loved one was generally the strongest fear for women and that fear of being punished by God ranked highest for men. Fear of snakes ranked sixth among women and twenty-first among men. On the basis of his data, he concluded that the hypothesized "prepared fears" can be more parsimoniously explained by "our shared experiences and cultural environment" (p. 149). In a sense, the present findings substantiate Kirkpatrick's position. They suggest that the hypothesis of prepared fear of survival-relevant cues may not be the most fruitful addition to the Pavlovian theory of fear acquisition. Rather, it may turn out that both the nonrandomness of fears and the most important experimental data on prepared learning (Öhman, 1986) can be explained by adding the notion of spatiotemporal unpredictability to Pavlovian theory. That is, given a certain aversive stimulus (UCS) and certain probabilistic associations between the occurrence/nonoccurrence of CS and UCS, with all other variables held constant, the spatiotemporal unpredictability that subjects attribute to the CS is positively related to the state of extinction. This formulation has a number of testable implications. Meanwhile it is acknowledged that problems in replicating and cross-validating the Öhman results (McNally, 1987; Merckelbach *et al.*, 1988) are not clarified by the present data. It might be worthwhile, however, to examine post hoc whether,

in successful replications of the Öhman results, the neutral and phobia-relevant CS are more divergent in terms of spatiotemporal unpredictability than the unsuccessful replications.

In the same vein as Bennett-Levy and Marteau (1984), who suggested that it is responses to perceptual characteristics rather than complete stimulus templates that have become genetically coded, it may, of course, be argued that the readiness to become fearful of unpredictable cues, is something which evolved through natural selection (Merckelbach, van den Hout, & van der Molen, 1987). The adaptational significance of sensitivity to unpredictable events seems self-evident. Plausibility aside, however, this notion will be hard to prove. It may be more rewarding to examine the merits of the present formulation by means of habituation (e.g., the match/mismatch paradigm introduced by Rachman & Lopatka, 1986) and conditioning procedures. From the present perspective, it would be most relevant to test whether induced fear responses to unpredictable CS are, indeed, quickly acquired and slowly extinguished.

4.3 Are prepared fears less severe, but more resistant to treatment?

Summary—In order to investigate the relationship between the extent to which clinical fears are prepared and clinically relevant characteristics of these fears (i.e. severity, age of onset and treatment outcome), the records of 63 obsessional and phobic patients were examined. Four independent raters scored the usable records ($N = 55$) on preparedness. The preparedness scores were combined and related to objective indices of severity (patients' scores on the Fear Survey Schedule, the Zung Depression Scale and the Maudsley Obsessional–Compulsive Inventory, as well as the treatment duration), onset ages and treatment outcomes (pre-treatment minus post-treatment MOCI scores, for a subsample of obsessional patients only). In contrast to earlier studies, it was found that product–moment correlations among preparedness ratings were relatively low and that prepared fears did not make up a majority in the sample. Indices of severity either did not correlate at all or correlated negatively with preparedness ratings. The positive correlation between preparedness and onset ages reached borderline significance. Evidence suggestive of a resistance to treatment of prepared fears was obtained.

INTRODUCTION

According to Seligman (1971), most human phobias are based on a genetic disposition or preparedness to associate fear with those objects and situations (e.g. snakes, enclosed places) that were threatening to our prehistorical ancestors.

Seligman's proposal is widely seen as an important contribution to the learning theory of anxiety (De Horne, 1980; Eysenck, 1982; Mineka, 1986). This is due in part to the psychophysiological studies of Öhman and co-workers. Using electric shock as an aversive stimulus, they conditioned the electrodermal response of normal *S*s to either prepared (slides of snakes and spiders) or neutral (slides of mushrooms and flowers) stimuli in a number of studies. They consistently demonstrated that the electrodermal response, once conditioned, extinguishes more slowly to prepared than to neutral stimuli [see the review by Öhman (1986)].

Because prepared learning is said to be biological and non-cognitive in nature, several authors have suggested that prepared fears are easily acquired and resistant to extinction (Seligman, 1971; Rachman and Seligman, 1976). More specifically, one would expect prepared phobias and obsessions to be more severe and to be characterized by poor treatment outcome and early age of onset.

Although two retrospective studies reported that prepared fears made up a majority of those fears found in a sample of clinical phobias and obsessions, they failed to find significant correlations between preparedness and clinically relevant characteristics (e.g. severity, age of onset and treatment outcome) of simple phobias and obsessive–compulsive disorders (de Silva, Rachman and Seligman, 1977; Zafiropoulou and McPherson, 1986). However, in both studies, most of the clinically relevant variables were evaluated by therapists.

Since the concept of preparedness is of potential value to clinicians (Sturgis and Scott, 1984), it was decided to carry out a correlational analysis of the association between the extent to which a clinical fear is rated as prepared and more objectively defined indices of the severity and outcome of these fears. In addition, the relationship between preparedness and age of onset was examined.

METHOD

Patients

The records of 50 obsessive–compulsive patients and 13 patients with simple phobias who had been referred to the Behavior Therapy Unit at Vijverdal Hospital in Maastricht over a 2-yr period were examined. Sixteen patients were out-patients and 47 were in-patients. Their mean age was 29 yr (range: 16–77 yr). There were 39 females (62%) and 24 males (38%) in the sample. Eight patients (13%) suffered from severe rituals not associated with a feared stimulus and consequently, the cases of these patients were not scored in terms of preparedness. In the records of the remaining 55 patients (87%), specific stimuli that gave rise to intensive anxiety could be identified. Some patients feared several stimuli; in a number of cases, the feared stimulus was identical for several patients. All in all, short descriptions of 55 separate objects and situations associated with anxiety were extracted from the records. Three behaviour therapists and one child psychologist, none of whom were acquainted with the preparedness hypothesis but rather with a basic knowledge of Darwinian theory, rated the content of these 55 times on preparedness, using the 5-point scale (1 indicating 'least prepared' and 5 indicating 'most prepared') introduced by de Silva *et al.* (1977). The raters had not been involved in the diagnosis

or in the treatment of the patients whose fears were rated. As de Silva *et al.* (1977) obtained a low agreement between raters when the behaviour of patients was rated on preparedness, raters were asked to rate only the content of the fears on preparedness.

Clinically relevant variables

Since it is part of the normal procedure at the Behavior Therapy Unit Vijverdal that patients fill out the Fear Survey Schedule (FSS; Wolpe and Lang, 1964), the Maudsley Obsessional–Compulsive Inventory (MOCI: Rachman and Hodgson, 1980) and the Zung Self-rating Depression Scale (SDS; Zung, 1965) at the beginning of their treatment, relatively objective indices of the severity of the patients' complaints were available for a large number of cases. In addition, most of the records provided detailed information about the number of previous psychotherapeutic treatments that the patients had undergone in relation to his/her complaints (in the form of a 4-fold classification: 0 = no previous treatment; 1 = one previous treatment; 2 = two previous treatments; 3 = three or more previous treatments), the age of onset of the complaints (in terms of years), and the treatment duration (in terms of weeks).

MOCIs were completed only by patients with a diagnosis of obsessive–compulsive disorder, whereas the SDS and the FSS were completed by both phobic and obsessional patients. The total MOCI score of each obsessional patient was derived by expressing the summed subscale scores of the patient as a percentage of the maximum obsessional score (30). Twenty-two obsessional patients (35%) completed the MOCI a second time at the end of their treatment. The differences between MOCI scores at the beginning and those at the end of the treatment were taken as an index of treatment outcome for this subsample. The patients in this subsample were treated with behavioural techniques, mainly exposure and response prevention.

RESULTS

Relatively low product–moment correlations between the preparedness ratings of the four raters were obtained. Correlations varied from 0.43 to 0.76, but were all significant ($N = 55$, $P < 0.05$, one-tailed). The mean correlation, after Fisher's r to z transformations (McNemar, 1969), was 0.59 ($N = 55$, $P < 0.05$, one-tailed).

The preparedness ratings of the four raters were summed and related to the corresponding records. If a patient's phobia pertained to more than one discrete stimulus, the summed preparedness ratings for the stimuli in question were averaged. The distribution of the usable cases ($N = 55$) along the preparedness continuum is shown in Fig. 1. A Kolmogorov–Smirnov one-sample test (Siegel, 1956) showed that the distribution did not deviate significantly from a normal distribution ($D = 0.13$, $N = 55$, $P < 0.27$, two tailed). The mean preparedness score was 9.3 (SD = 3.9), which is low compared to the mean scores reported by de Silva *et al.* (1977) and Zafiropoulou and McPherson (1986) (12.6 and 14.4, respectively, when their mean scores are transformed into the present format). Thirty-three (78%) of the 55 cases had a preparedness score that was $\leqslant 12$. It should be noted that a score of 12 in the present study corresponds to a rating of 3 in the original de Silva *et al.* scale. According to the instructions a rating of 3 is to be given to "objects or situations that were *possibly* dangerous to pretechnological man under some *uncommon* circumstances" (de Silva *et al.*, 1977, p.67; italics added).

Product–moment correlations between summed preparedness ratings and clinical variables are shown in Table 1. Due to missing data in the records, the sample size varies from correlation to correlation. As for the relationship between preparedness and severity, no significant correlations in the expected direction were found. Interestingly enough, the negative association between preparedness and MOCI scores ($r = -0.42$, $N = 40$, $P < 0.05$, two-tailed) was significant, whereas the negative correlation of preparedness with the number of previous treatments reached borderline significance ($r = -0.25$, $N = 55$, $0.05 < P < 0.10$, two-tailed).

As far as the association between preparedness and ages of onset is concerned, a correlation contrary to that which was expected was found: the higher the preparedness score the later the age of onset ($r = 0.29$, $N = 45$, $0.05 < P < 0.10$, two-tailed).

For 17 (27%) of the 22 obsessional patients who completed the MOCI at the beginning (MOCI-1) and at the end (MOCI-2) of the treatment preparedness ratings were available. A negative correlation between preparedness and treatment outcome in terms of MOCI change scores was found ($r = -0.61$, $N = 17$, $P < 0.05$, one-tailed), which is in line with the expectation that prepared fears are resistant to extinction. As preparedness was negatively associated with the severity of the disorder, it could be argued that the negative association between preparedness and treatment outcome reflects a floor effect. In order to investigate this possibility, the 17 cases were divided into two groups on the basis of the median preparedness score for this subsample. The 8 in-patients with a preparedness score $\leqslant 7$ (median score) formed the unprepared group. The prepared group consisted of the 3 out- and 6 in-patients with a preparedness score > 7. A Fisher exact probability test (Siegel, 1956) showed that the groups did not differ significantly in their respective frequencies of in- and out-patients ($P = 0.12$). Table 2 shows mean pre- and post-treatment MOCI percentages of the total obsessional score

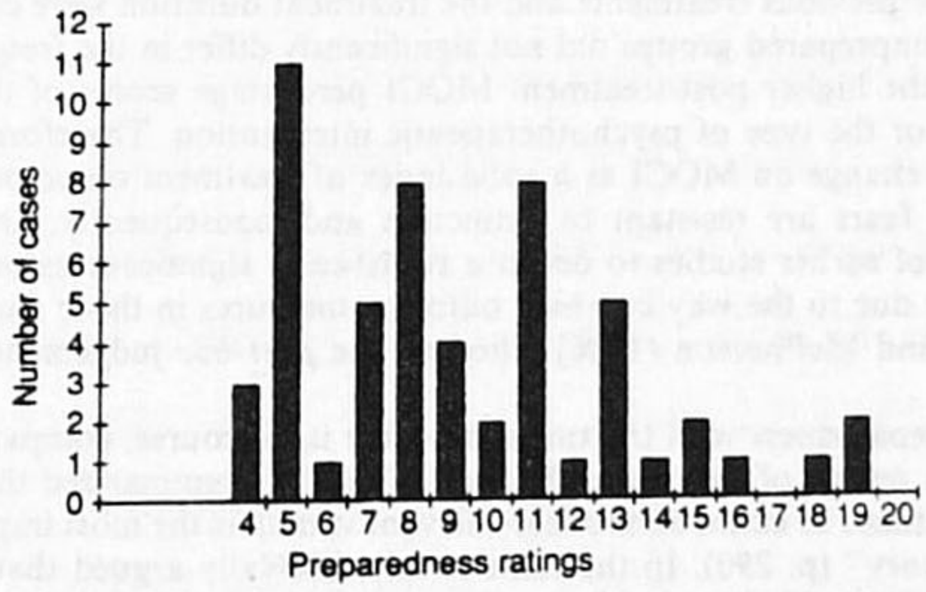


Fig. 1. Distribution of phobic and obsessional cases ($N = 55$) along the preparedness continuum. The preparedness ratings presented here are the sum of separate ratings of four raters.

Table 1. Product-moment correlations between preparedness ratings and patients' scores on the various measures

Measure	Preparedness[a]
FSS	−0.05 (46)
MOCI-1	−0.42* (40)
SDS	−0.08 (49)
Treatment duration	−0.04 (55)
Number of previous treatments	−0.25** (55)
Age of onset	0.29** (45)
MOCI-1 − MOCI-2	−0.61* (17)

The sample size is given in parentheses. Since a number of correlations were in directions opposite to those predicted, two-tailed *P*-values are given.

[a]Sum of the separate ratings of four raters.

*$P < 0.05$. **$0.05 < P < 0.10$.

Table 2. Mean percentages of the total obsessionality score on the pre-treatment MOCI-1 and the post-treatment MOCI-2 of the prepared ($N = 9$) and unprepared ($N = 8$) groups of obsessional patients

	MOCI-1	MOCI-2
Unprepared group ($N = 8$)	69.3 (14.9)	19.3 (17.9)
Prepared group ($N = 9$)	53.8 (13.1)	37.0 (13.6)

Standard deviations are given in parentheses.

for the period and unprepared group. As can readily be seen, a cross-over rather than a floor effect occurred after treatment. A 2 (prepared vs unprepared group) × 2 (pre- vs post-treatment MOCI percentages) with a repeated measure as the last factor, did not reveal a main group effect. The main effect of treatment, however, was significant [$F(1,15) = 59.2$, $P < 0.05$], as was the interaction effect of group × treatment [$F(1,15) = 14.6$, $P < 0.05$] caused by the greater change on the MOCI of the unprepared group after treatment. The significant interaction of group × treatment did not disappear when treatment duration or number of previous treatments was used as covariates.

Finally, it should be noted that the correlations among the clinical variables, were, on the whole, in the expected direction and that a number of them reached significance. For example, the pre-treatment MOCI percentage scores correlated positively with the number of previous treatments ($r = 0.37$, $N = 46$, $P < 0.05$, one-tailed), FSS scores ($r = 0.34$, $N = 41$, $P < 0.05$, one-tailed) and SDS scores ($r = 0.32$, $N = 45$, $P < 0.05$, one tailed) and correlated negatively with ages on onset ($r = -0.24$, $N = 43$, $0.05 < P < 0.10$, one-tailed). As a result, there was no reason to question the coherence of the data used in this study.

DISCUSSION

Compared to the results reported by de Silva *et al.* (1977) and Zafiropoulou and McPherson (1986), relatively low correlations between the preparedness ratings of independent raters were obtained. The low correlations were probably caused by the fact that the raters had to base their judgements on rather short descriptions of the clinical cases. In contrast to the earlier studies, we were unable to show that the majority of phobias and obsessions in our sample could be rated as prepared. It may well be that this finding is caused by the fact that our raters, in contrast to the raters in the de Silva *et al.* study (1977), were unfamiliar with the preparedness hypothesis.

No evidence was found in support of the predicted, positive relationship between preparedness and severity. In fact, analysis revealed a negative correlation between preparedness and pre-treatment MOCI percentage scores that reached significance. Furthermore, there was a tendency for preparedness to be negatively associated with the number of previous psychotherapeutic treatments. Even though the idea of a positive relationship between preparedness and severity at first seems to be plausible on a theoretical level, close inspection of the preparedness concept as formulated by Seligman (1971) reveals that this idea is in no way logically implied by the concept. In a paper entitled "Unprepared phobias: 'be prepared' ", Rachman and Seligman (1976) suggested that "those few people whose unprepared phobias last long enough and are severe enough to be seen in the clinic may be severely disturbed people or may have abnormal personalities" (p. 338). It must be recognized, therefore, that our failure to detect a positive association between preparedness and severity cannot be taken as an invalidation of the preparedness concept itself.

A prediction that flows directly from the preparedness concept is that prepared fears should be more easily acquired. Thus, we anticipated a negative correlation between ages of onset and preparedness. Our results did not substantiate this prediction. In this respect, they are consistent with the results reported by de Silva *et al.* (1977) and Zafiropoulou and McPherson (1986). As the positive correlation between preparedness and ages of onset was small and reached only borderline significance, we are reluctant to invoke alternative theoretical explanations.

As far as the relationship between preparedness and treatment outcome is concerned, a negative correlation between preparedness ratings and MOCI change scores was observed. A subsequent analysis of variance further clarified this finding. Obsessional patients whose fears were rated as relatively high on preparedness ended up after treatment with higher total MOCI scores as compared to patients whose fears were rated as relatively unprepared. This difference did not disappear when the effects of the number of previous treatments and the treatment duration were cancelled out in covariate analyses. Furthermore, the prepared and unprepared groups did not significantly differ in the frequency of in- and out-patients. We have no reason to believe that the higher post-treatment MOCI percentage scores of the prepared patients were caused by variables such as mediation, or the type of psychotherapeutic intervention. Therefore, to the extent that one is willing to accept pre- to post-treatment change on MOCI as a valid index of treatment outcome (Rachman and Hodgson, 1980), our data suggest that prepared fears are resistant to extinction and, consequently, are associated with relatively poor treatment outcome. The failure of earlier studies to detect a statistically significant association between preparedness and poor treatment outcome may be due to the way in which outcome measures in those studies were obtained. Both de Silva *et al.* (1977) and Zafiropoulou and McPherson (1986) relied on the *post-hoc* judgements of therapists for their outcome measures.

The negative association of preparedness with treatment outcome is, of course, compatible with the psychophysiological research on preparedness. In his review of this research, McNally (1987) summarized the experimental results as follows: "In any event, the enhanced resistance to extinction to fear-relevant stimuli is the most important experimental phenomenon consistent with preparedness theory" (p. 290). In the same article, McNally argued that "this laboratory phenomenon is the strongest support for a theory that holds that fears of evolutionary significance are extremely resistant to extinction when the clinical data suggest that they are not" (p. 298). The studies cited in support of this conclusion, however, were not designed to test the hypothesized association between preparedness and poor treatment outcome. Bearing in mind the

positive evidence found in this study, McNally's conclusion might be premature. Admittedly, the small sample size, the relatively low agreement between the preparedness ratings of the independent raters, and the absence of exact information regarding the therapeutic interventions all weaken the weight that can be attached to this positive evidence. To settle this issue, further studies investigating the relationship between preparedness and treatment outcome are clearly called for. Future research should preferably be based on a large sample and on detailed information concerning the therapeutic treatment.

Acknowledgements—The help received from Julien Peters and Eric Griez is gratefully acknowledged.

4.4 Psychophysiological and subjective reactions to facial stimuli in social phobics and normals

Summary

Nine social phobics and 9 normal control subjects were exposed to slides of angry faces, happy faces, and neutral objects (e.g., flowers or mushrooms). Skin conductance responses (SCRs) to the stimuli and eyeblink rate (EBR) during stimulus exposure were recorded. In addition, subjects were asked to rate the stimuli in terms of pleasantness. While angry face stimuli elicited greater SCRs, stronger inhibition of EBR, and were evaluated more negatively than the other stimuli, there were no differences between social phobics and normals in these respects. Thus, the findings lend no support to the idea that social phobics are particularly sensitive to facial cues in general or to negative facial cues in particular.

Key Words: social phobia, preparedness, SCR, EBR.

Introduction

In a series of studies, Dimberg (1982, 1986a, 1987) has explored the influence of different types of facial stimuli on psychophysiological responding and conditioning. Dimberg (1982) reported that stimuli depicting angry facial expressions and those depicting happy facial expressions elicit completely different patterns of electromyographic activity in normal subjects. Whereas the former class of stimuli induced more corrugator activity, which is associated with negative facial expressions, the latter class of stimuli induced more zygomatic activity, which is associated with positive facial expressions. Dimberg (1985) also showed that skin conductance responses (SCRs) conditioned to angry facial stimuli extinguish more slowly than SCRs conditioned to happy facial stimuli. A similar effect was obtained for cardiovascular reactions (Dimberg, 1987).

Dimberg (1987; Öhman & Dimberg, 1978) related his findings to the concept of "preparedness" formulated by Seligman (1971). According to

this concept, humans are biologically prepared to respond to those stimuli that were once threatening to prehistoric man. Seligman (1971) argued that the idea of biological preparedness would explain why most phobias pertain to a relatively narrow class of stimuli (e.g., snakes, high places, thunder, etc.). In a similar vein, Dimberg (1987) reasoned that humans are predisposed to react to threatening social cues (e.g., angry faces).

In their theoretical papers, Öhman and Dimberg (Öhman & Dimberg, 1984; Öhman, Dimberg & Öst, 1985; Öhman, 1986) have claimed that their experimental results sustain an interpretation of social phobias in terms of preparedness. This claim is based largely on the idea that the fear of facial cues plays a decisive role in social fears. For example, Öhman et al. (1985) suggested that "social fears are the result of a biologically determined readiness to easily associate fear to certain classes of stimuli, **and perhaps particularly those originating in the face"** (p. 31; italics ours). Those aspects of the face that are most likely to be prepotent are the eyes. First, fear and escape are induced by staring eyes and eye-like shapes in birds (Blest, 1957), chickens, and non-human primates (Redican, 1975). A wide range of species actually employ displays of eyes or eye-like spots to frighten others (Marks, 1987). Second, in normal humans, heart rate and skin conductance activity are higher when gazes are directed at, rather than away from, them (Ellsworth, 1975). Dimberg and Öhman (1983) found that extinction of SCRs conditioned to angry face stimuli is only delayed when the angry face is directed at, rather than turned away from, the experimental subject. Referring to these findings, Marks (1987) suggested that social phobics' fear of being watched is an exaggeration of the normal human sensitivity to eyes.

While the idea that social phobics are particularly responsive to angry facial expressions corresponds with clinical impressions (Marks, 1969, 1987), it has, to the present authors' knowledge, not been subjected to experimental tests. Dimberg, Frederikson, and Lundquist (1986; cited by Öhman et al., 1985) reported that subjects with high social fear gave significantly larger SCRs to social stimuli than subjects with low social fear. Yet, this study has two obvious limitations. First, it is not clear to what extent the social fear subjects

resembled social phobics. Second, the social stimuli used in the Dimberg et al. study (1986) consisted of neutral facial expressions.

The present study sought to ascertain whether social phobics are more responsive to angry facial expressions than normal control subjects. SCRs to, and subjective evaluation of, facial and non-facial (neutral) stimuli were measured in social phobics and normal controls. In addition, endogenous eyeblink rates (EBRs) were recorded during stimulus exposure. As it has been shown that visual tasks requiring attention inhibit EBRs (Stern, Walrath & Goldstein, 1984), it was expected that angry facial cues would inhibit EBRs more than happy facial or neutral cues, and especially so in social phobics.

Method

Subjects

Eighteen subjects (4 men and 14 women) participated in the experiment. Their mean age was 22 years (range 18-31).

Social phobics (N=9: 2 men and 7 women) were recruited by an advertisement placed in both a local newspaper and a university magazine. The advertisement described social phobia symptoms (e.g., shyness, tension in social situations, etc.) and invited readers with these symptoms to contact the researchers by telephone. Persons who responded were first screened in an interview that took place over the phone. They were later asked to come to our laboratory and to complete several questionnaires, among them the fear questionnaire (FQ; Marks & Mathews, 1979). Subjects with a primary diagnosis of depression, agoraphobia, or simple phobia were excluded from the experiment. Of the remaining subjects with a primary diagnosis of social phobia, one used minor tranquilizers. None of the subjects was undergoing any kind of behavioral treatment at the time.

Control subjects (N=9: 2 men and 7 women) were recruited from undergraduate psychology courses. The control group was matched on age, sex, and educational level. Control subjects were also invited to complete the FQ.

Apparatus and Stimulus Materials

During the experiment, psychophysiological and subjective responses to angry facial expressions, happy facial expressions, and neutral stimuli were recorded.

A number of psychophysiological responses, namely skin conductance levels (SCLs), SCRs, EBRs, and respiration rate (RR), were measured. SCLs and SCRs were recorded using two Beckman Ag-AgCL electrodes (8 mm diameter) attached to the medial phalanges of the second and third fingers of the right hand. The electrodes were connected to a Beckman Skin Conductance Coupler (type 9844).

EBRs were derived from the vertical electro-oculogram. Two Beckman Ag-AgCL electrodes, one placed two cm above the right eye and one placed two cm below the right eye, were dc coupled (Beckman Voltage Coupler), with the low-pass filter set at 50 Hz.

RR was recorded using a Beckman Respiration Belt connected to a Beckman Voltage/Pulse/Pressure Coupler (type 9853A).

SCRs, EBRs, and RRs were recorded on a Beckman R 711 polygraph (paper speed 5 mm/sec).

Stimuli were scored in terms of "pleasantness", using a 10 cm visual analog scale, with -5 indicating "extremely pleasant", 0 indicating "neutral", and +5 indicating "extremely unpleasant".

All subjects saw one slide of an angry face, one slide of a happy face, and one non-facial, neutral slide (e.g., either a flower or a mushroom). The facial stimuli were taken from Ekman and Friesen (1975). All gazes from the facial cues were directed at the subject.

The slides were projected onto a white screen, 2 m in front of the subject. The size of the projected image was approximately 75 x 110 cm. A Kodak Carousel was used for stimulus presentation.

A PDP Minc 11 Microcomputer controlled stimulus presentation and registration of psychophysiological responses.

Design

The experiment was conducted according to a 2 (group) x 3 (stimulus type) design, with the first factor being a between-subject factor

(social phobics vs. normal controls) and the second factor being a within-subject factor (angry face; happy face; neutral stimulus).

Subjects saw not only facial stimuli but also a non-facial, neutral stimulus in order to control for the possibility that social phobics react more strongly to facial cues (friendly and happy faces) **per se**.

Each slide was presented to the subject 6 times, and consequently, a trial factor, containing 6 levels, was included in the statistical analysis.

Procedure

Subjects sat in a comfortable chair that was placed in a dimly lit, sound-attenuated chamber. The apparatus was located in an adjacent room. The stimuli were projected through a hole in the wall.

The subjects were told that the experiment dealt with subjective and physiological reactions to facial and non-facial cues. They were instructed to watch the stimuli and to avoid unnecessary movements. After the subjects had given their consent and recording sites had been cleaned with distilled water, electrodes were attached with adhesive collars. A 4-minute resting period then followed, during which baseline values of SCL and EBR were obtained. The slides were then presented to the subjects in a random order. The only restriction was that no more than two successive slides of one type (e.g., angry face) would be shown. Slide duration was 8 sec. The intertrial interval varied from 15 to 21 sec, with a mean of 18 sec.

In order to correct for the influence of irrelevant stimulus details, different subjects within a given group saw different people exhibiting the facial expressions and different neutral stimuli. However, for each subject, the angry face stimulus, happy face stimulus, and neutral stimulus were kept constant across trials.

At the end of the experiment, the three stimuli were presented once more, and the subject was asked to rate the stimulus in terms of pleasantness.

Data Reduction and Analysis

t-Tests were used to evaluate group differences in fear questionnaire scores and in baseline SCL and EBR.

SCRs to the stimuli were defined as the maximal deflection occurring 1 to 8 sec after stimulus onset. SCR and SCL values were measured in micromho and square root-transformed. RR was recorded as a control variable. That is, trials with respiratory irregularities were excluded from the data analysis. Such irregularities occurred on less than 3% of all trials. SCR values for these trials were estimated on the basis of the adjacent trials.

As for EBR, the total number of eyeblinks between stimulus onset and offset were counted. Both SCR and EBR values were subjected to 2 (group) x 3 (stimulus type) x 6 (trials) analysis of variance (ANOVA) with repeated measures on the last two factors. For effects involving a trial factor, Greenhouse-Geisser corrected probability levels were used.

Results

FQ-Scores and Psychophysiological Baseline Values

As is evident from Table 1, the social phobia group and the control group did not differ in FQ-agoraphobia scores or FQ-blood/injury scores. The groups also had comparable baseline values for SCL and EBR. The only notable difference between the groups appeared on the FQ-social phobia scale, with social phobics scoring significantly higher than normal controls. Thus, it can safely be concluded that the social phobia group was characterized specifically by social fears, rather than by an elevated level of generalized fear. The mean FQ-social phobia score of the social phobics came close to 4, which corresponds to "would definitively avoid it" (Marks & Mathews, 1979).

Table 1. MEAN FQ SUBSCALE SCORES (AGORAPHOBIA, BLOOD/INJURY PHOBIA, AND SOCIAL PHOBIA) AND SQUARE ROOTED BASAL CONDUCTANCE (SCL) AND EYEBLINK RATE (EBR; PER MINUTE) DURING THE BASELINE PERIOD FOR THE SOCIAL PHOBIA AND CONTROL GROUP. STANDARD DEVIATIONS ARE GIVEN IN PARENTHESES.

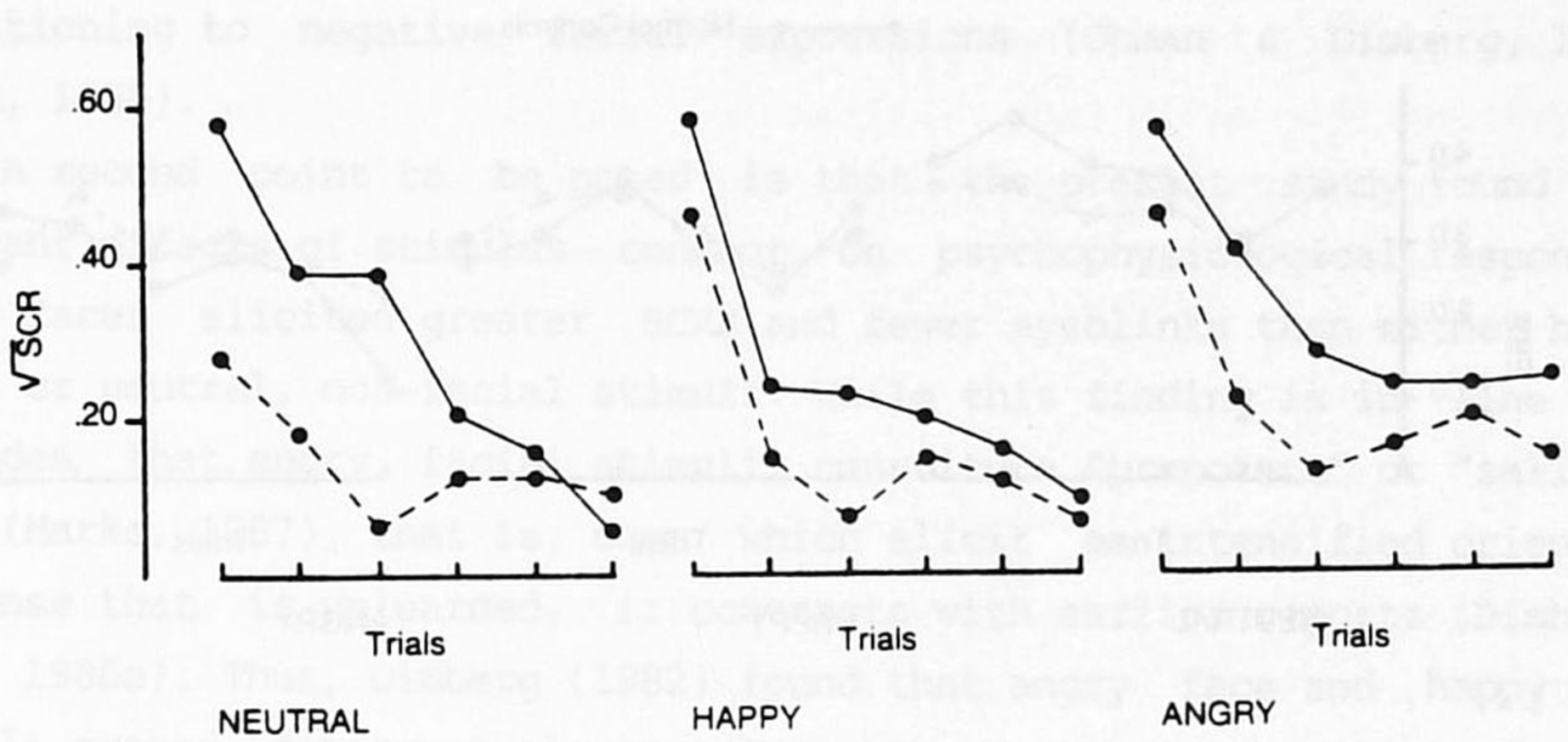
	FQ-agoraphobia	FQ-blood/ injury phobia	FQ-social phobia	SCL	EBR
Social Phobics	1.0 (1.4)	1.6 (1.4)	3.5* (1.7)	2.1 (0.7)	20.0 (6.4)
Normal Controls	0.6 (0.9)	1.8 (1.0)	1.9 (1.5)	2.0 (0.5)	16.5 (8.2)

*$p<0.05$, d.f.=16, one-tailed

SCRs to Facial and Non-Facial Stimuli

Figure 1 shows SCRs to non-facial, neutral stimuli (left panel), happy face stimuli (middle panel), and angry face stimuli (right panel). A 2 (groups) x 3 (stimulustype) x 6 (trials) ANOVA revealed a significant main effect of trials [$F(5,80) = 15.0$, $p< 0.05$], due to an overall response decrement over trials. Furthermore, a significant main effect of stimulus type was found [$F(2,32) = 4.0$, $p<0.05$]. This effect was due to the fact that angry face stimuli elicited greater SCRs than either happy face stimuli or neutral stimuli. Post hoc t-tests confirmed that responses to angry face stimuli were larger than those to neutral stimuli [$t(17)=1.6$, $p=0.06$, one-tailed] or to happy face stimuli [$t(17)=3.5$, $p<0.05$, one-tailed], with means ($\sqrt{SCR}$) of 0.28 (s.d.=0.22), 0.23 (s.d.=0.22), and 0.21 (s.d.=0.18), respectively. However, no significant main effect of group [$F(1,16)=1.4$, $p=0.26$] or interaction effect of group with stimulus type [$F(2,32)<1$] was found. Thus, social phobics and normal controls were not found to be different in their SCRs to facial stimuli.

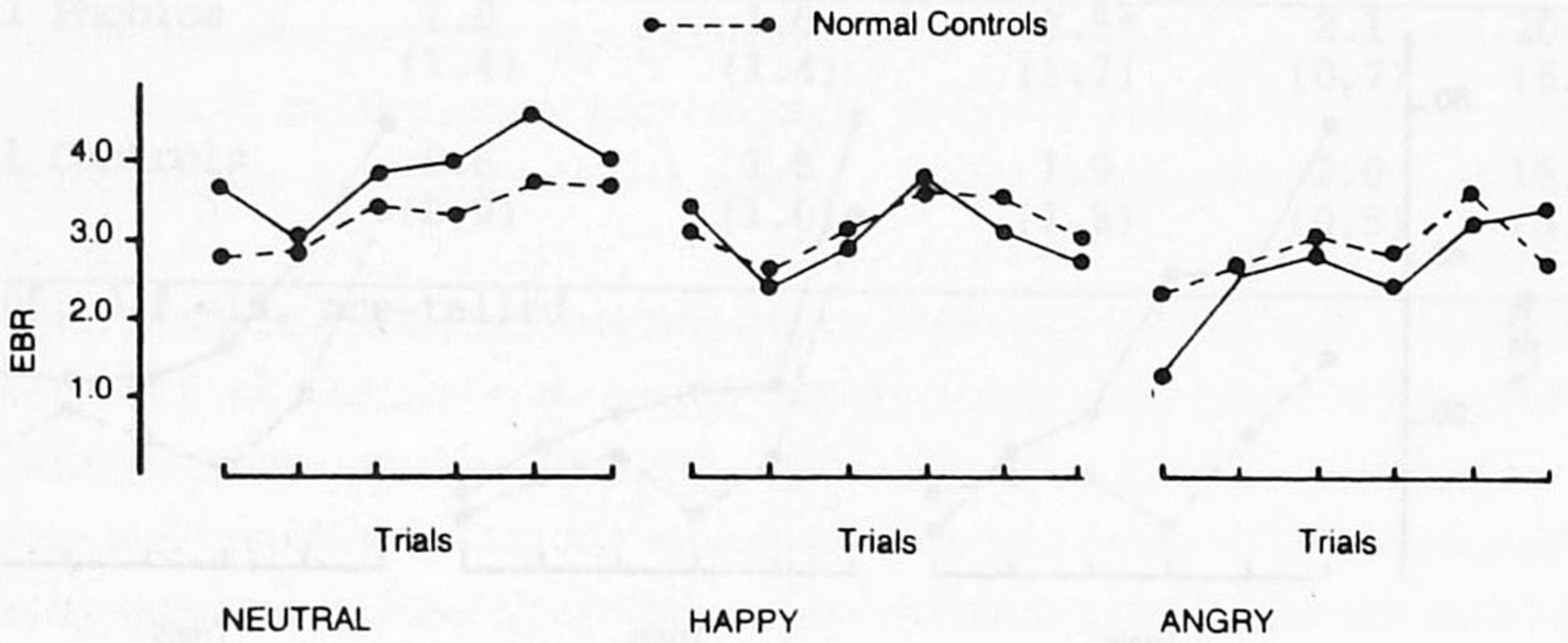
Figure 1. MEAN √SCRS AS A FUNCTION OF TRIALS (6) TO NON-FACIAL, NEUTRAL STIMULI (LEFT PANEL), HAPPY FACE STIMULI (MIDDLE PANEL) AND ANGRY FACE STIMULI (RIGHT PANEL) OF THE SOCIAL PHOBIA AND CONTROL GROUP.



EBRs during Facial and Non-Facial Stimuli

Mean EBRs during presentation of neutral stimuli (left panel), happy face stimuli (middle panel), and angry face stimuli (right panel) are shown in Figure 2. A 2 (group) x 3 (stimulus type) x 6 (trials) ANOVA revealed a borderline significant effect of trials [$F(5,80)=2.5$, $p=0.08$], caused by the slight increase in EBR over trials. Furthermore, a significant main effect of stimulus type emerged [$F(2,32)=6.2$, $p<0.05$]. Post hoc t-tests indicated that the EBR during presentation of angry face stimuli was lower than the EBR during presentation of happy face stimuli [$t(17)=1.5$, $p=0.08$, one-tailed], while the EBR during presentation of happy face stimuli was significantly lower than that during presentation of neutral stimuli [$t(17)=2.0$, $p<0.05$, one-tailed], the means being 2.8 (s.d.=1.7), 3.1 (s.d.=1.6), and 3.6 (s.d.=1.9), respectively. Again, no significant differences were found between social phobics and normals.

Figure 2. MEAN EBR AS A FUNCTION OF TRIALS (6) DURING NON-FACIAL, NEUTRAL STIMULI (LEFT PANEL), HAPPY FACE STIMULI (MIDDLE PANEL), AND ANGRY FACE STIMULI (RIGHT PANEL) OF THE SOCIAL PHOBIA AND CONTROL GROUPS.



Subjective Evaluation of Facial and Non-Facial Stimuli

At the end of the experiment, subjects were asked to rate the stimuli in terms of pleasantness (-5 - +5). A 2 (groups)x 3 (stimulus type) ANOVA revealed no significant group differences. The only effect reaching significance was a main effect of stimulus type [$F(2,32)=12.8$ $p<0.05$]. Post hoc t-tests showed that angry faces were rated as significantly more negative than non-facial, neutral stimuli [$t(17)=4.6$, $p<0.05$, one-tailed], while these latter stimuli were rated as relatively more negative than happy faces [$t(17)=2.5$, $p<0.05$, one-tailed]. The means were +4.0 (s.d.=2.9), 0.5 (s.d.=1.4), and -2.0 (s.d.=2.5), respectively (positive values indicate negative evaluation).

Discussion

If angry facial expressions were to function as primary conditioned stimuli in social phobics (Öhman, 1986), one would predict stronger responses to these cues in social phobics than in normal controls, and if

it were human eyes in particular that social phobics were hypersensitive to, then social phobics and normals would respond differently to facial vs. non-facial stimuli. The present study failed to substantiate either of these predictions. No significant difference was found between social phobics and normal controls for either the psychophysiological or the subjective data. Consequently, the current findings run counter to the idea that social phobias can be understood as the outcome of prepared conditioning to negative facial expressions (Öhman & Dimberg, 1984; Öhman, 1986).

A second point to be noted is that the present study found significant effects of stimulus content on psychophysiological responses. Angry faces elicited greater SCRs and fewer eyeblinks than either happy faces or neutral, non-facial stimuli. While this finding is in line with the idea that angry facial stimuli constitute "prepotent" or "salient" cues (Marks, 1987), that is, cues which elicit an intensified orienting response that is unlearned, it contrasts with earlier reports (Dimberg, 1982, 1986a). Thus, Dimberg (1982) found that angry face and happy face stimuli evoked divergent electromyographic patterns, yet elicited equal SCR magnitudes in normal subjects. Similarly, Dimberg (1986a; exp.1 through exp.4) found no differences in SCRs to angry and happy face stimuli during the habituation phase of his conditioning experiments. It is unclear why the present study succeeded in finding SCR differences for angry and happy face stimuli whereas Dimberg failed to observe such differentiation. However, the fact that differential effects of stimulus content were not restricted to the SCR mode but also appeared for EBR underlines the validity of the present results.

As far as EBR is concerned, some remarks are in order. Solid evidence has indicated that endogenous eyeblinks are associated with a partial loss of information intake (Wittenmeyer, Stern & Chen, 1983). It has also been shown that the greater the attentional demands of a task, the greater the inhibition of EBR (see review by Stern et al., 1984). Given the reduction in visual information processing associated with each eyeblink, the inhibition of EBR during a difficult visual task would seem to be adaptive (Stern et al., 1984). The reduced EBR during exposure to angry face stimuli reported above suggests that the attentional demands of such stimuli are greater than those of happy face or non-facial,

neutral stimuli. The gradual increase in EBRs over trials that was found in the present study can best be explained as a time-on-task effect. Several experiments have demonstrated that repetitive tasks that lead to boredom or fatigue are accompanied by an increase in EBR (Stern et al., 1984).

In sum, the present study found that angry face stimuli elicit greater SCR magnitudes and fewer eyeblinks than do either happy face stimuli or non-facial, neutral stimuli. This finding is in line with the suggestion that angry face stimuli are salient, prepotent cues (Marks, 1987). Yet, no evidence was found to suggest that facial stimuli in general, or angry face stimuli in particular, are more salient for social phobics than for normal controls.

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We gratefully acknowledge the technical advice regarding eyeblinks that we received from Dr. John A. Stern (Washington University).

4.5 Electrodermal conditioning to stimuli of evolutionary significance: Failure to replicate the preparedness effect

In the present study, an attempt was made to replicate the preparedness effect reported by Öhman, Fredrickson, Hugdahl, & Rimmö (1976). Following Öhman et al. (1976) as closely as possible, a differential conditioning procedure was carried out in which subjects' skin conductance responses (SCRs) were conditioned either to stimuli of evolutionary significance (slides of snakes and spiders) or to evolutionally neutral stimuli (slides of mushrooms and flowers). The experiment consisted of 8 habituation, 12 acquisition, and 20 extinction trials. Electric shock served as an unconditioned stimulus during the acquisition phase. Although SCRs showed significant decreases during habituation and were significantly influenced by the conditioning procedure during acquisition, they were not found to extinguish significantly more slowly in the group that saw slides of snakes and spiders. This result contradicts the earlier results reported by Öman and colleagues. Possible explanations for this failure to replicate their results are discussed.

KEY WORDS: preparedness; classical conditioning; skin conductance; phobias.

INTRODUCTION

To account for the selectivity with which certain stimuli (e.g., snakes, spiders, closed spaces, etc.) elicit phobic reactions, Seligman (1971) introduced

the concept of preparedness. According to this concept, stimuli that have been of critical importance to survival in the course of human evolution can enter more readily into association with aversive, unconditioned stimuli. The concept of preparedness is, therefore, based on a combination of Darwinian and Pavlovian notions.

The clinical studies of De Silva, Rachman, and Seligman (1977) and Zafiropoulou and McPherson (1986) demonstrated, indeed, that most simple phobias and obsessive-compulsive disorders pertain to evolutionally significant objects and situations. Both studies can be regarded as indirect support for the preparedness hypothesis. However, the most impressive and convincing evidence for this hypothesis to date has come from the experimental work of Öhman and co-workers. Using electric shock as an unconditioned stimulus (UCS) and slides of snakes and spiders or mushrooms and flowers as conditioned stimuli (CS), they found in more than a dozen studies that the skin conductance response (SCR), once conditioned, extinguishes more slowly to evolutionally significant (i.e., snakes and spiders) than to evolutionally neutral (i.e., mushrooms and flowers) stimuli (for reviews see Öhman, 1986; Öhman *et al.* 1976; Öhman, Fredrikson, & Hugdahl, 1978; Öhman; Dimberg, & Öst, 1985).

Although the preparedness effect (i.e., the selective resistance of the SCR to extinction in the case of evolutionally significant stimuli) apparently occurs consistently in the differential conditioning paradigm used by Öhman and colleagues, there is some reason to question the reliability of this phenomenon. First, researchers using different conditioning procedures did not find evidence for a preparedness effect (Deitz, 1982; Emerson & Lucas, 1981; McNally, 1981, 1986; McNally & Reiss, 1982, 1984; Vaitl, Gruppe, & Kimmel, 1985).

Second, outside the Öhman group, only Cook, Hodes, and Lang (1986, experiments 5 and 6), using a differential conditioning paradigm, claimed to have replicated the preparedness effect. But close inspection of their data reveals that the effect these authors found was rather weak; in one experiment (5) it fell short of the conventional confidence level ($p < 0.05$) and in the other experiment its significance was dependent on the type of SCR measurement (probability vs. magnitude). Meanwhile, at least four unsuccessful attempts to replicate the preparedness effect are known (Eelen, cited by Emmelkamp, 1982; Cook, 1981, and Hodes, 1981, both cited by Cook *et al.*, 1986; McNally & Foa, 1986), but unfortunately, only one of these attempts (McNally & Foa, 1986) has been published in extensive form. However, in making comparisons one must bear in mind that the McNally and Foa study differs from the procedure of Öhman *et al.* (1976) in two important ways. First, while Öhman and associates worked with unselected subjects, the subjects in the McNally and Foa study were deliberately selected on the

basis of their extreme scores on an animal fear questionnaire. It is conceivable that in the groups with initially high fear a generalized stress effect, due to the phobic slides, occurred and, consequently, obscured differential extinction. Lumsden, Howard, and Fenton (1986), studying contingent negative variation in phobic volunteers, reported a similar effect. The second procedural difference is that, in order to cancel out the influence of irrelevant background features of the slides, Öhman and colleagues showed different slides to different subjects in the same group. McNally and Foa failed to do this.

In summary, studies carried out to cross-validate the preparedness effect yielded conflicting results. Given the potentially great value of the preparedness concept for the development of sophisticated theories of learning in general (cf. discussions regarding the equipotentiality premise given by Seligman and Hager, 1972) and for experimental models for simple (Öhman *et al.*, 1978) as well as social (Dimberg, 1986) phobias in particular, further tests of the reliability of the preparedness effect are called for. With this in mind, the authors conducted a study following the conditioning procedure of Öhman *et al.* (1976) as closely as possible. In a differential conditioning paradigm, subjects were conditioned either to slides of snakes and spiders (evolutionally significant group) or to slides of mushrooms and flowers (evolutionally neutral group). The hypothesis tested was that the conditioned SCR would show more resistance to extinction with the former type of stimuli used as the CS than with the latter.

METHOD

Subjects

The subjects were 42 undergraduate students (10 males and 32 females) with no current or prior history of phobic complaints. Their mean age was 21.7 years, with a range from 17 to 35 years. Subjects were paid for their participation in the study.

Apparatus and Stimulus Materials

SCR and skin conductance level (SCL) were recorded with a Beckman skin conductance coupler (Type 9844), using the method of constant voltage (.5 V). The coupler allowed for a maximum sensitivity of .05 μmho. After cleaning each subject's fingers with 70% alcohol, Beckman Ag–AgCl electrodes (diameter, 8 mm), filled with Hewlett Packard Redux paste, were at-

tached with adhesive collars to the medial phalanx of the second and third fingers of the left hand. An electric stimulator with a maximum capacity of 40 mA delivered an electric current (dc) to the subjects. Two shock electrodes were placed on the first finger of the subject's left hand. A Kodak Carousel was used for presentation of the slides. Onset and offset of the stimuli, intertrial intervals, occurrence of the electric pulses, and response registration were controlled by a microcomputer (PDP Minc-11). The slides were projected onto a white wall. The size of the projected image was approximately 80 × 120 cm, 2.5 m in front of the subject.

Design

A 2 (group) × 2 (conditioning) factorial design with repeated measures on the last factor was used. For statistical analyses, a trial factor, in the form of a repeated measure, was added. The group factor refers to the type of slides the subjects saw. The conditioning factor is a consequence of the fact that each subject saw two slides, one of which (CS+) was associated with electric shock (UCS) and the other (CS−) never being followed by an UCS.

Procedure

Upon arrival in the laboratory, subjects were asked to sit down in a comfortable chair which was placed in a dimly lit, sound-attenuated chamber. The recording apparatus, microcomputer, and Kodak Carousel were in an adjacent room. The slides were projected through a hole in the wall. Before the actual experiment began, subjects filled out two questionnaires. The first questionnaire asked for fear ratings on the animal items of the FSS (Wolpe & Lang, 1964; Macdonough, 1976). The second questionnaire (Amsterdamse Biografische Vragenlijst; Wilde, 1962) was the Dutch equivalent of the Eysenck Personality Inventory (Eysenck & Eysenck, 1964) and measured neuroticism and extraversion. The experimenter then explained that during the experiment electric shocks would occur. After subjects had given their consent and electrodes had been fastened, the experimenter started a shock workup procedure in which the shock level was gradually increased until the subject indicated that the shock was "uncomfortable but not painful." Subjects were not instructed about the CS–UCS contingency.

The experiment consisted of three phases. The first was a habituation procedure. This phase involved eight CS-only trials (four CS+ and four CS–). An acquisition phase then followed, in which six reinforced presentations of CS+ and six unreinforced presentations of CS− occurred. Finally, there was an extinction phase, consisting of 10 unreinforced presentations

of both slides. Slides were presented for 8 sec. The duration of the shock was .5 sec and it was delivered exactly upon the removal of CS+. Intertrial intervals varied between 20 and 40 sec, in steps of 5 sec, and with a mean of 30 sec. Throughout the experiment, the order of presentation of the two slides was quasi-random; no more than two successive presentations of the same slide occurred. Subjects were randomly assigned to one of the two groups. The conditioning factor was counterbalanced so that, for example, in the group that saw evolutionally significant slides, snakes and spiders served an equal number of times as CS+. To randomize out irrelevant background features of the pictures, the subject saw the same two slides during the experiment, but different subjects within the same group saw different slides.

Response Definition and Analysis

SCLs were measured on four occasions during the experiment: at the very beginning of the habituation phase, between habituation and acquisition, between acquisition and extinction, and finally, at the end of the extinction phase. The experimental procedure allowed for the recordings of multiple response forms of the SCR (Prokasy & Kumpfer, 1973). Like Öhman *et al.* (1976), differentiations were made between FAR, SAR, and TOR components of the SCR. The FAR (first-interval anticipatory response) pertains to a maximal deflection with a latency of 1–4 sec after CS onset. The SAR (second-interval anticipatory response) is defined as a maximal deflection at 4–8 sec after CS onset. Maximal deflections occurring at 1–4 sec after CS offset during the habituation and extinction phases are regarded as TORs (third-interval omission responses). The SCL and the SCR components were measured in micromhos and square-root transformed. Data were analyzed as response magnitudes.

Using the *t* statistic, group differences in mean shock level, neuroticism, extraversion, FSS scores, and initial SCL were examined. The course of the SCLs of the two groups throughout the experiment was investigated with an analysis of variance. Separate analyses of variance were carried out for FARs, SARs, and TORs during the three phases of the experiment. In addition, separate *t* tests for FARs, SARs, and TORs on successive CS+ and CS− extinction trials were carried out. The rejection region for all comparisons was set at $p < 0.05$.

RESULTS

One female subject in the evolutionally neutral group showed extremely high SCLs. Consequently, SCR recordings were unreliable in this case.

Table I. Mean Square-Root SCL for the Evolutionally Significant and Evolutionally Neutral Groups During Four Successive Stages of the Experiment[a]

	A		B		C		D	
Evolutionally significant	3.5	(.73)	3.5	(.77)	3.6	(.81)	3.1	(1.04)
Evolutionally neutral	3.8	(.79)	3.9	(.91)	4.1	(.86)	3.8	(1.01)

[a]A, before habituation; B, after habituation; C, after acquisition; D, after extinction. Standard deviations are given in parentheses.

Her record was excluded from further analysis, leaving 19 subjects in the evolutionally neutral and 22 subjects in the evolutionally significant group. The mean of the UCS level was 9.8 mA in the evolutionally significant group and 8.9 mA in the evolutionally neutral group. This difference did not reach significance. There were also no significant differences in neuroticism, extraversion, FSS scores, or initial SCL between the groups. A 2 (group) × 4 (occasions) analysis of variance, with a repeated measure as its last factor, was carried out on the square-root basal conductance data (see Table I). Although there was no main effect for groups, differences between the two groups did emerge during the experiment, as suggested by a significant group × occasions interaction [$F(3, 117) = 2.8, p < 0.05$].

FAR

There was a significant main effect of trials during habituation [$F(3, 117) = 6.6, p < 0.05$], indicating a decrease in FARs with trials (left panels, Fig. 1). In addition, a significant conditioning × trials interaction was found [$F(3, 117) = 5.4, p < 0.05$]; during the first trials the CS− elicited a greater response than the CS+, but this difference disappeared during the later habituation trials. No other effects reached significance in this phase.

During acquisition (middle panels, Fig. 1), no main effect of conditioning occurred. Furthermore, the group × conditioning interaction did not reach significance, which means that there was no difference in ease of acquisition between the groups. However, as a significant conditioning × trials interaction [$F(5, 195) = 8.2, p < 0.05$] made clear, the conditioning procedure was not ineffective; while responding to CS− trials showed a progressive decrease, the FARs to CS+ first increased or remained stable and then decreased.

During extinction, the effects of conditioning disappeared quickly (right panels, Fig. 1). Both the main effect of conditioning and the group × conditioning interaction (i.e., the preparedness effect) failed to reach significance. The only source that did reach significance in this phase was a main effect of trials [$F(9, 351) = 5.0, p < 0.05$]. As is apparent from Fig. 1, this was due to a general decrease in FARs over trials. Separate *t* tests revealed that,

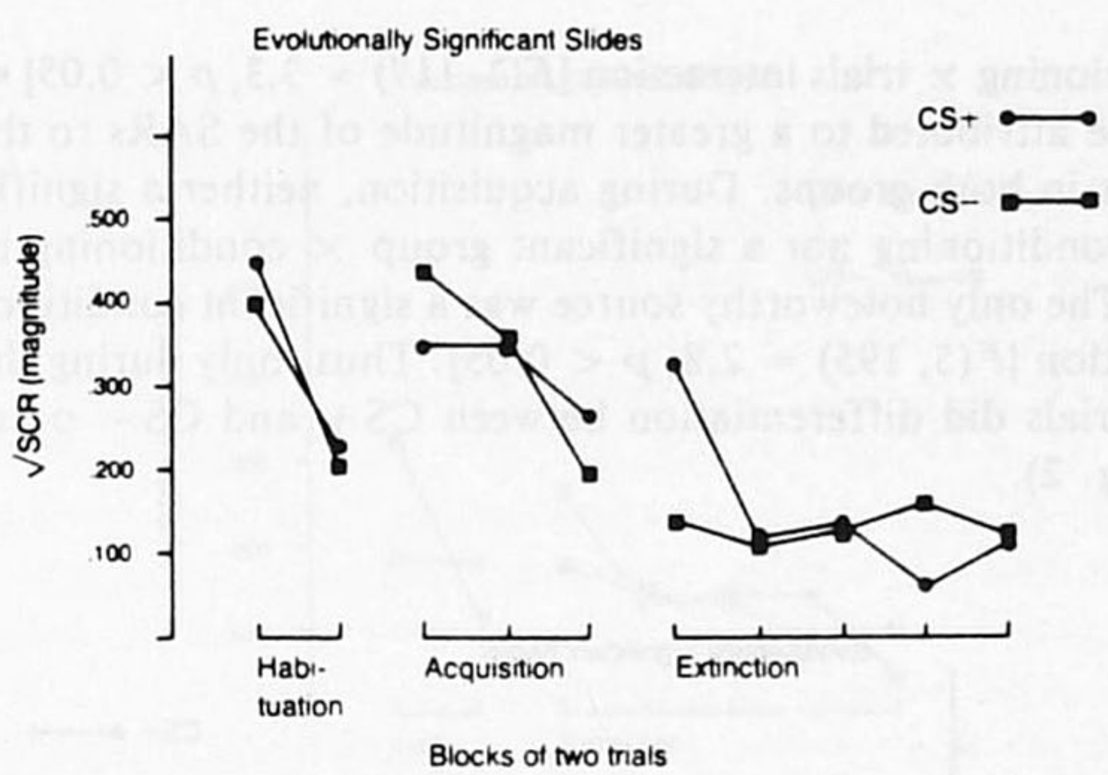

Fig. 1. Mean magnitude √SCR plotted as a function of trial blocks for the first-interval anticipatory response (FAR) to reinforced (CS+) and nonreinforced (CS−) stimuli for the two groups.

except for a significant difference between the responses during the second CS+ and CS− extinction trials, the FARs of both groups showed no reliable differentiation between CS+ and CS−.

SAR

An analysis of variance of the SAR data from the habituation revealed a significant main effect of trials [$F(3, 117) = 7.1$, $p < 0.05$], indicating a decrease in responses across trials (left panels, Fig. 2). Moreover, a signifi-

cant conditioning × trials interaction [$F(3, 117) = 3.3, p < 0.05$] was found. This can be attributed to a greater magnitude of the SARs to the last two CS+ trials in both groups. During acquisition, neither a significant main effect of conditioning nor a significant group × conditioning interaction emerged. The only noteworthy source was a significant conditioning × trials interaction [$F(5, 195) = 2.8, p < 0.05$]. Thus, only during the later acquisition trials did differentiation between CS+ and CS− occur (middle panels, Fig. 2).

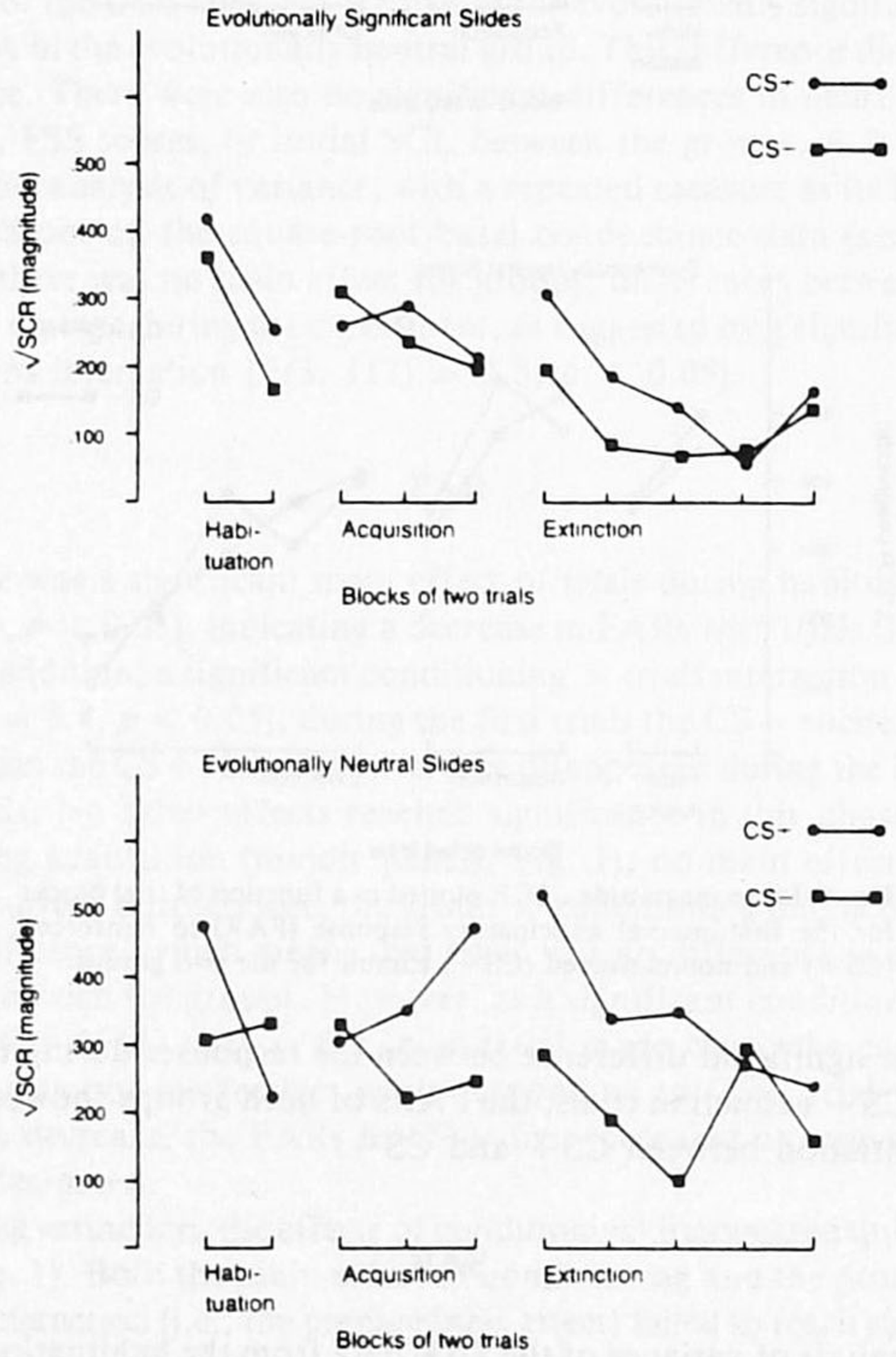

Fig. 2. Mean magnitude √ SCR plotted as a function of trial blocks for the second-interval anticipatory response (SAR) to reinforced (CS+) and nonreinforced (CS−) stimuli for the two groups.

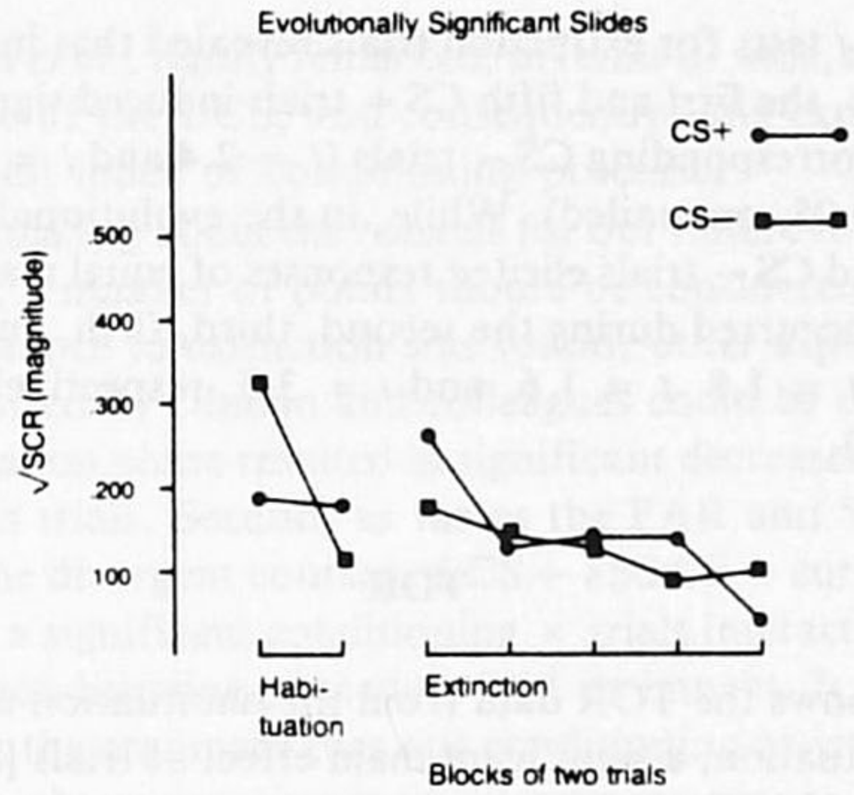

Fig. 3. Mean magnitude √ SCR plotted as a function of trial blocks for the third-interval omission response (TOR) to reinforced (CS+) and nonreinforced (CS−) stimuli for the two groups.

The most outstanding feature of the SAR data from the extinction (right panels, Fig. 2) was the appearance of a strong conditioning effect [$F(1, 39) = 14.5, p < 0.05$], indicating that, overall, reliable differentiation between CS+ and CS− occurred. Furthermore, a significant main effect of trials was found [$F(9, 351) = 5.4, p < 0.05$]. This effect resulted from the progressive decrease in SARs over trials in both groups. However, no evidence was found for a preparedness effect here either, since the group × conditioning interaction failed to reach significance.

Individual *t* tests for extinction trials revealed that in the evolutionally significant group, the first and fifth CS+ trials induced significantly stronger SARs than the corresponding CS− trials ($t = 2.4$ and $t = 2.0$, respectively; df = 21; $p < 0.05$, one tailed). While, in the evolutionally neutral group, the first CS+ and CS− trials elicited responses of equal magnitude, differential responding occurred during the second, third, fifth, and sixth extinction trials ($t = 3.0$, $t = 1.8$, $t = 1.6$, and $t = 3.5$, respectively; $df = 18$; $p < 0.05$, one tailed).

TOR

Figure 3 shows the TOR data from the habituation and extinction trials. During habituation, a significant main effect of trials [$F(3, 117) = 12.3$, $p < 0.05$] and a significant interaction of conditioning × trials [$F(3, 117) = 4.8$, $p < 0.05$] was found. The latter effect is the result of a steeper decrease in TORs during the CS− trials in both groups (left panels, Fig. 3). During extinction, a significant effect of trials emerged [$F(9, 351) = 4.6$, $p < 0.05$] due to the overall decrease in TORs over trials (right panels, Fig. 3). Moreover, a significant conditioning × trials interaction was found [$F(9, 351) = 2.7$, $p < 0.05$]. Separate *t* tests for the extinction trials showed that this effect was especially strong in the evolutionally neutral group. While, in the evolutionally significant group, even the first CS+ trials did not elicit superior responding, the evolutionally neutral group showed reliable differentiation for the first three pairs of trials ($t = 2.7$, $t = 2.9$, and $t = 2.1$, respectively; $df = 18$; $p < 0.05$, one tailed). However, it should be stressed that the analysis of variance yielded no evidence for a significant group × conditioning interaction.

DISCUSSION

The present study found no evidence to suggest that a resistance to extinction of the SCR was specific to the group that saw stimuli of evolutionary significance. This is in marked contrast to the results reported by Öhman and associates. They showed in several studies a preparedness effect which was especially manifest in the FAR and TOR, but less so, or not at all, in the SAR component of the SCR (Öhman *et al.*, 1976; Hugdahl, Fredrikson, & Öhman, 1977; Hugdahl & Öhman, 1980). It might, therefore, seem surprising that in our study the SAR component appeared in both groups to be the most sensitive measure for remaining effects of conditioning during extinction. On the other hand, as Gray (1982), in his critical review of the

studies by Öhman *et al.*, rightly remarked, in terms of time, the SAR is closest to the occurrence of the UCS, and consequently, one expects this component to be the best index of conditioning processes.

Before speculating about the reasons for our failure to replicate the preparedness effect, a number of points should be considered. First, although no selective resistance to extinction was found, other aspects of the conditioned SCR reported by Öhman and colleagues could be clearly replicated. Thus, the habituation phase resulted in significant decreases in FARs, SARs, and TORs across trials. Second, as far as the FAR and SAR components are concerned, the divergent courses of CS+ and CS− curves during acquisition resulted in a significant conditioning × trials interaction. This demonstrates that the conditioning procedure had its impact. It should be added immediately that the argument that our conditioning procedure was not extensive enough to observe preparedness phenomena cannot be taken as a serious objection. To begin with, prepared learning is, by definition, learning that occurs without extensive acquisition (Seligman & Hager, 1972). Moreover, Öhman, Eriksson, and Olofsson (1975) claimed that a single CS-UCS trial is sufficient to observe a preparedness effect.

Why, then, were we unable to replicate the preparedness effect? One possibility is that the stimuli we used were not salient enough. The slides we used were unambiguous, straight forward, clearly recognizable pictures of the objects in their natural environment. Thus, unless preparedness is a highly artificial phenomenon which depends on extremely salient stimuli, we think that lack of saliency is a possible, but not very satisfactory, explanation for our failure to observe preparedness.

We used alcohol rather than distilled water in our electrodermal preparation. Admittedly, this departs from customary procedure, but it seems an unlikely explanation for the absence of differential extinction in the two groups.

It is very unlikely that certain features of our samples are responsible for the absence of preparedness. As in the studies by Öhman and colleagues, our subjects were undergraduates and blind as to the purpose of the experiment. There was, indeed, a high proportion of females in our sample, but it should be remembered that Fredrikson, Hugdahl, and Öhman (1976) found no sex differences in the occurrence of prepared learning.

There were no striking group differences that could account for our failure to observe prepared learning; groups did not differ in neuroticism, extraversion, fear scores on animal items, or initial SCL. Differences in SCL between the two groups did appear during the experiment, yet we hesitate to attach much significance to this finding. While it is known that SCR and SCL correlate positively with one another (Martin & Rust, 1976), Hugdahl and Öhman (1980) reported a preparedness effect in spite of SCL differences

between their control and their experimental group. Moreover, in testing for a wide range of possible but unintended differences between experimental and control subjects, type 1 errors are rather likely. Nevertheless, a confounding influence of SCL on the occurrence of prepared learning cannot be ruled out.

Possibly of more importance than subject characteristics is the intensity of the UCS. It is conceivable that in our study, conditioning, in the sense of CS-UCS learning, took place but that the UCS was not sufficiently aversive. At the same time it seems reasonable to expect that with high-intensity shocks preparedness effects disappear, since powerful unconditioned stimuli can establish robust fear responses to any stimulus (e.g., Campbell *et al.*, 1964). To the best of our knowledge, data about the shock intensities Öhman and associates used during their experiments have not been published, so that a direct comparison is not possible. It should be stressed again that the procedure for determining shock intensities was identical to that of Öhman and co-workers.

In our instructions to the subjects, we did not inform them about the CS-UCS contingency. Giving subjects information about this contingency is not without consequences, as can be concluded from a study by Öhman *et al.* (1976). In their first experiment, they informed their subjects about the CS-UCS relationship, which resulted in an almost immediate, differential responding to CS+ and CS− during the habituation phase. Furthermore, Maltzman (1979) convincingly demonstrated that the SCR is very sensitive to information about "what leads to what" which subjects either receive from the experimenter or gather themselves. His finding is in line with the view of Dawson and Furedy (1976). These authors maintain that awareness of the CS-UCS relationship is a necessary condition for autonomic differential classical conditioning. This line of reasoning is not compatible with Seligman's (1971) assumption that prepared learning is noncognitive. On the other hand, it should be noted that Öhman *et al.* (1976, experiment 3) reported a preparedness effect in a group that had not been instructed about the CS-UCS relationship. Nonetheless, in view of the Maltzman (1979) study, we feel that a systematic investigation of the influence of instruction on the occurrence of prepared learning in a differential conditioning procedure is important.

In the present study we have relied on skin conductance as an indicator of sympathic activity. However, recent research by Cook *et al.* (1986) suggests that cardiovascular responding may provide a more reliable and successful index in preparedness studies (but see also Fredrikson & Öhman, 1979).

To summarize, we can only speculate about the reasons for our failure to observe a preparedness effect. The intensity of the UCS, the influence that instructions have on conditioning, and the type of psychophysiological

variables that are measured may offer some explanation and, therefore, deserve closer attention in future investigations. In light of the numerous studies by Öhman and associates that show clear-cut preparedness phenomena, our failure might seem puzzling. However, as previously mentioned, neither investigators using a different conditioning (Deitz, 1982; Emerson & Lucas, 1981; McNally, 1981, 1986; McNally & Reiss, 1982; Vaitl *et al.*, 1985) nor McNally and Foa (1986), following a differential conditioning paradigm, were able to demonstrate preparedness. Future research should, therefore, concentrate on the critical conditions for the occurrence of prepared learning. Unless we can identify these conditions, we will not be in a position to determine the degree to which experimentally induced preparedness is a valuable laboratory model for simple and social phobias.

ACKNOWLEDGMENTS

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4.6 Electrodermal and cardiovascular responses to phobia-relevant stimuli in a trace conditioning paradigm: Effects of instruction

Keywords: preparedness, classical conditioning, instructions, skin conductance, heart period, phobias.

SUMMARY The present study concerns itself with the hypothesis that the preparedness phenomenon demonstrated by Öhman and co-workers (1985) is non-cognitive in nature, i.e., is not dependent upon subjects deliberately focusing their attention on the contingency between a phobia-relevant conditioned stimulus (CS+) and shock. In order to test this hypothesis, a trace conditioning experiment was carried out in which one group of subjects saw a phobia-relevant CS+ and a second group saw a neutral CS+. During acquisition, the former group was confronted with the sequence phobia-relevant CS+ (slides of snakes or spiders)/shock/non-reinforced, neutral CS− (slides of mushrooms or flowers), while the latter group was confronted with the sequence neutral CS+/shock/phobia-relevant CS−. Within each group, half of the subjects received instructions that stressed the CS+/shock contingency ('forward' instruction), while the other half was asked to pay special attention to the CS−, which always followed shock ('backward' instruction). The experiment consisted of 6 habituation, 12 acquisition, and 24 extinction trials. Both skin conductance responses (SCRs) and heart rate responses (HRRs) were measured. SCRs were found to extinguish more slowly in subjects who had received a 'forward' instruction than in subjects with a 'backward' instruction. However, there was no resistance to extinction of the conditioned SCRs in subjects who saw a phobia-relevant CS+ and received a 'backward' instruction. Thus, the delayed extinction of conditioned SCRs was not dependent upon the phobia relevance of the CS+, but rather upon the amount of attention that subjects were asked to pay to the CS+/shock contingency. For HRRs, a less comprehensive picture emerged, with phobia-relevant CS+s eliciting accelerative responding during habituation, a small deceleration during acquisition, and no significant changes from baseline during extinction. Results are related to other recent studies that have yielded contradictory evidence for the alleged non-cognitive nature of the preparedness phenomenon.

Introduction

Seligman's preparedness hypothesis (1971) assumes that fear of certain situations and objects (e.g. enclosed spaces, snakes) once contributed to the survival chances of our ancestors and, consequently, became genetically coded in the form of a primitive learning mechanism. Seligman holds that this mechanism, when activated by mild, aversive events, very easily associates fear with these formerly survival-threatening objects and situations. The existence of such a mechanism would explain why most phobias pertain to a relatively limited class of stimuli (De Silva, Rachman, and Seligman, 1977; Zafiropoulou and McPherson, 1986). In addition, the biological and primitive nature of the hypothesized mechanism would offer an explanation for the irrational, non-cognitive nature of phobic anxiety.

Results reported by Öhman and co-workers

have generally been taken as strong support for the preparedness hypothesis (see Eysenck, 1982; Sturgis and Scott, 1984; Mineka, 1985). Using electric shock as an aversive, unconditioned stimulus by means of which the skin conductance response (SCR) of normal subjects was conditioned either to slides of snakes and spiders (phobia-relevant stimuli) or to slides of mushrooms and flowers (neutral stimuli), Öhman and co-workers demonstrated in more than a dozen studies that the conditioned SCR extinguishes more slowly to phobia-relevant stimuli than to neutral stimuli (see reviews by Öhman, Dimberg, and Öst, 1985; Öhman, 1986; McNally, 1987). Öhman and associates argued that this delayed extinction with phobia-relevant stimuli documents the selectivity of phobic cues that results from prepared learning. Yet, as Seligman's hypothesis implies that prepared learning is non-cognitive, an issue that warrants consideration is the relationship between cognitive processing and the phenomenon found by the Öhman group. More specifically, the question emerges of whether the occurrence of this phenomenon depends upon the salience of the contingency between a particular slide and electric shock.

The few studies that directly touch upon the role of cognitions in the acquisition and extinction of prepared associations have produced mixed results (see review of Merckelbach, van der Molen, and van den Hout, in press). Both Hugdahl and Öhman (1977) and Hugdahl (1978) found that the delayed extinction of the conditioned SCR to phobia-relevant slides occurs even when subjects are instructed that no more shocks will be delivered. They interpret this finding as 'an analogy to the irrationality of phobic fears' (Hugdahl, 1978; p. 321). However, recent research casts some doubt on this interpretation and on the empirical facts upon which it is based. First, Cook, Hodes, and Lang (1986), as well as McNally (1981), were unable to replicate the 'delayed extinction despite instruction' phenomenon. Second, Dawson, Schell, and Tweddle Banis (1986) found no evidence for the acquisition of a conditioned SCR to phobia-relevant CS+s in subjects who were unaware of the contingency between CS+ and shock. They carried out a conditioning experiment that was embedded in a masking task, making it relatively difficult for the subject to become aware of the contingencies between slides and shocks. The subject's awareness of these contingencies was measured by having the subject express his expectancy of the shock on a trial-by-trial basis throughout the acquisition and extinction phases of the experiment. Interestingly, in those subjects who eventually became aware of the critical contingency, the decrease in electrodermal responding to phobia-relevant CS+s during extinction mirrored the decline in expectancy of a shock. But, as Dawson *et al.* admitted, their procedure differed markedly from that typically followed in the Öhman experiments. In particular, the verbal and non-verbal feedback of the subjects might have interfered with the conditioning and subsequent extinction of the SCR.

The present experiment addressed the question as to whether an effect of phobia relevance on the extinction of conditioned autonomic responses can be demonstrated when both the conditioning procedure and the instructions given to the subjects reduce the salience of the contingency between slide and shock. To enhance compatibility with the research of the Öhman group, a trace conditioning procedure (Prokasy and Kumpfer, 1973), quite similar to one of the techniques used by Hugdahl and Öhman (1980), was employed. Trace conditioning was preferred, since the time gap between offset of the CS+ and occurrence of a shock prevents subjects from becoming immediately aware of the contingency. In order to further influence the salience of the CS+/shock contingency, the subjects' perception of this contingency was manipulated by instructions. Half of the subjects were told that the CS+ would frequently precede shock, whereas the other half was asked to concentrate on the stimulus that followed shock (CS−). The hypothesis tested was that the latter set of instructions favours a quick extinction of conditioned autonomic responses to CS+ presentations, except when the CS+ is phobia-relevant. If the phenomenon reported by Öhman and colleagues is, indeed, an example of non-cognitive prepared learning, delayed extinction of the conditioned responses to a phobia-relevant CS+ should occur, despite the relatively reduced salience of the CS+/shock relationship.

Cook *et al.* (1986) recently suggested that cardiovascular responses are much more sensi-

tive to effects of phobia relevance than are SCRs. They found, during the acquisition phase of their experiments, a robust acceleration that was specific for cardiac responses to phobia-relevant CS+s. Cook *et al.* interpreted this cardiac acceleration as a 'defensive response' (Graham and Clifton, 1966). In light of the Cook *et al.* findings, the present study employed both electrodermal and cardiac measures. In addition, subjects were asked, at the end of the experiment, to evaluate the CS+ and CS− slides in terms of pleasantness. This was done in order to examine whether subjective evaluations of CSs were affected by instructions concerning the CS/shock relationship. As has been demonstrated by the extensive research of Martin and Levey (1978), the evaluative response is sensitive to various aspects of the conditioning process.

Method

Subjects

Fifty-two undergraduate students (44 women and 8 men) with no current or prior history of phobic complaints volunteered to participate in the study. Their mean age was 22 years (range: 17–38 years). They were recruited from an introductory psychology course. The subjects were paid for their participation. They were told that the experiment was an investigation of the effects of visual and tactile stimuli on the SCR.

Apparatus, stimulus materials, and assessment

SCR and skin conductance level (SCL) were measured with a Beckman Skin Conductance Coupler (type 9844), using the method of constant voltage (0.5 volts). The coupler allowed for a maximum sensitivity of 0.05 micromho. Electrodes were attached with adhesive collars to the medial phalanges of the second and third fingers of the subject's left hand. Heart rate responses (HRRs) were recorded from electrodes arranged according to a lead II placement (Stern, Ray, and Davis, 1980) and connected to a Beckman Voltage/Pressure/Volume Coupler (type 9853 A). For both HRR and SCR/SCL recordings, Beckman Ag-AgCl electrodes (8 mm diameter) filled with Hewlett Packard Redux paste were used. SCR recording sites were cleaned with distilled water, whereas HRR recording sites were cleaned with 70 per cent alcohol. SCR and HRR were continuously monitored by a Beckman Polygraph (type R 611).

An electric stimulator with a maximum capacity of 40 mA delivered an electric current (dc) to the subject. Two shock electrodes were attached to the first finger of the subject's left hand.

A microcomputer (PDP Minc-11) controlled onset and offset of the stimuli, inter-trial intervals, occurrence of the shock, and response registration. The microcomputer was also used for on-line processing of the interbeat (R-R) intervals.

Slides were presented with a Kodak Carousel and projected onto a white wall, 2.5 m in front of the subject. The size of the projected image was approximately 80 × 120 cm. The slides were straight-forward, clearly recognizable representations of snakes, spiders, mushrooms, and flowers.

After the experimental procedure, subjects rated the 'pleasantness' of the slides on a 10 cm visual analog scale, which ranged from 0 ('rather pleasant') to 10 ('extremely unpleasant').

Design

The experiment was designed according to a 2 (stimulus) × 2 (instruction) × 2 (reinforcement) factorial model, with repeated measures on the last factor. Thus, the experiment comprised four conditions. In the statistical analysis, a trial factor, in the form of a repeated measure, was added.

The stimulus factor refers to the sequence of the stimuli during the acquisition phase of the experiment. For one group of subjects, shock was preceded by a phobia-relevant stimulus (slide of a snake or a spider) and followed by a neutral stimulus (slide of a mushroom or a flower). The other group was confronted with the reversed stimulus order. As all subjects saw one neutral and one phobia-relevant slide, the present procedure differs from the differential conditioning procedure typically used in the Ohman studies. We refrained from using the differential conditioning procedure typically employed by Öhman and associates because it has been suggested that this set-up might interfere with the effects of phobia relevance on the magnitude of the conditioned response during acquisition (McNally, 1987).

The instruction factor pertains to the two different sets of instructions that were given. Within each group, half of the subjects received a 'forward' instruction. That is, subjects were informed that, during the experiment, one and the same slide would frequently precede a shock. The other half of the subjects were given a 'backward' instruction. In the 'backward' condition the shock was depicted as a warning stimulus that signalled the occurrence of a subsequent CS−. Subjects receiving the 'backward' instruction were asked to pay close attention to the details of this CS− 'in order to be able to perform a recognition task at the end of the experiment'.

The reinforcement factor is a consequence of the fact that each subject saw two slides, one of which (CS+) was followed by a shock during the acquisition phase of the experiment and the other (CS−) which was never followed by a shock.

Procedure

Subjects were invited to sit down in a comfortable chair which was placed in a soundproof, electrically shielded, and dimly lit chamber. The recording apparatus, microcomputer, and Kodak Carousel were placed in an adjacent room. Slides were projected through an opening in the wall. The experimenter explained that shocks would occur during the experiment. Once subjects had given their consent and recording sites had been cleaned, electrodes were attached. A shock work-up procedure followed in which the shock level was gradually increased until the subject indicated that the shock was 'rather unpleasant but not painful'. Subjects then received either 'forward' or 'backward' instructions. In neither condition were subjects told exactly how often shocks would occur.

The experiment consisted of three parts. The first was a habituation phase which involved 6 CS only trials (3 CS+ and 3 CS−). Then, 6 acquisition series followed, each series consisting of the sequence CS+/shock/CS−. Two non-reinforced CS+ and 2 additional CS− trials were dispersed among these series (see below). Finally, subjects received 24 unreinforced extinction trials (12 CS+ and 12 CS−).

Slides were presented for 4 sec. The duration of the shocks was 0.5 sec. Shocks were administered during the acquisition phase, exactly halfway between the CS+ and CS− inter-trial interval, making the procedure a form of trace conditioning. Throughout the experiment, inter-trial intervals varied between 12 and 24 sec, with a mean of 18 sec.

The order in which the two slides were presented was quasi-random during habituation and extinction: no more than two successive presentations of the same slide occurred. Throughout the acquisition phase of the experiment, shock was always preceded by a CS+ and followed by a CS− slide. However, to maintain an impression of random stimulus order, 2 non-reinforced CS+ slides and 2 additional CS− slides were presented, making the acquisition phase a 75 per cent reinforcement schedule.

After the extinction trials, the CS+ and CS− slides were presented one more time, and subjects were asked to evaluate both slides in terms of pleasantness.

Subjects were randomly assigned to one of the four experimental groups (phobia-relevant CS+/'forward' instruction, phobia-relevant CS+/'backward' instruction, neutral CS+/'forward' instruction and neutral CS+/'backward' instruction), each group containing 13 subjects.

Data reduction and analysis

SCL was measured on four occasions during the experiment: at the beginning of the habituation phase, between habituation and acquisition, between acquisition and extinction, and, finally, at the end of the extinction phase. SCR was defined as the maximal deflection occurring 1–4 sec after CS onset. Both SCL and SCR were measured in micromho. For reasons discussed by Levey (1980), SCL and SCR were square foot transformed; this was done prior to any statistical analysis. HRR was expressed in terms of heart period (msec per beat) and analyzed according to the computational procedure proposed by Graham (1978b). Interbeat intervals were recorded for the last pre-stimulus beat and six successive beats occurring after stimulus onset. To obtain beat-by-beat cardiac waveforms, interbeat intervals after stimulus onset were subtracted from the pre-stimulus baseline. With this procedure, positive values indicate acceleration, whereas negative values reflect deceleration. Using univariate analyses of variance

Table 1 Mean square-root SCL during four successive stages of the experiment[a].

	occasions			
groups	A	B	C	D
1	3.2 (0.85)	3.6 (0.87)	3.7 (0.97)	3.8 (1.19)
2	2.9 (0.49)	3.2 (0.59)	3.3 (0.66)	3.2 (0.71)
3	3.1 (0.60)	3.4 (0.74)	3.5 (0.75)	3.3 (0.68)
4	2.9 (0.59)	3.0 (0.76)	3.2 (0.68)	3.2 (0.84)

[a]1, the phobia-relevant CS+/'forward' instruction group; 2, the phobia-relevant CS+/'backward' instruction group; 3, the neutral CS+/'forward' instruction group; 4, the neutral CS+/'backward' instruction group. A, before habituation; B, after habituation; C, after acquisition; D, after extinction.

(ANOVAs) group differences in mean shock level, SCL and subjective evaluation of the slides were examined. To analyze SCR and HRR, separate ANOVAs were carried out for the habituation, acquisition, and extinction phases of the experiment. A rejection region of $p < 0.05$ was adopted for all tests. Effects involving large degrees of freedom due to repeated measures were evaluated with Greenhouse–Geisser corrected probability levels. If not indicated otherwise, Greenhouse–Geisser conservative probabilities were used for these effects.

Results

Shock intensity, SCL, and subjective evaluation

A one-way ANOVA indicated that the groups did not differ in mean shock intensities [$F(3,48) < 1.00$]. Averaged over all subjects, the shock intensity was 15.6 mA (SD = 11.4). A 2 (stimulus) × 2 (instruction) × 4 (occasions) ANOVA, with the last factor being a repeated measure, and which was performed on the square root SCL data, showed that there were no group differences here either. The only source reaching significance was a main effect of occasions [$F(3,144) = 16.40$, $p < 0.05$], caused by a general, linear increase in SCL during the experiment (Table 1).

Mean pleasantness ratings for CS+ and CS− slides, which were obtained at the end of the experiment, are given in Table 2. Due to procedural errors, the ratings of 7 subjects were not usable. As is evident from the data presented in Table 2, the 'forward' instruction was associated with a more negative evaluation of the CS+, whereas the 'backward' instruction was associated with a more negative evaluation of the CS−. To examine the effects of instruction on stimulus evaluation, the CS+ and CS− evaluative ratings were subjected to separate 2 (stimulus) × 2 (instruction) ANOVAs. For the CS+ data, a main effect of stimulus [$F(1,44) = 13.08$, $p < 0.05$] and a main effect of instruction [$F(1,44) = 6.40$, $p < 0.05$] was found. The interaction of stimulus with instruction remained non-significant [$F(1,44) = 2.02$, $p = 0.16$]. Similar results were obtained for the CS− data. Thus, the main effect of stimulus [$F(1,44) = 10.81$, $p < 0.05$] and of instruction [$F(1,44) = 7.71$, $p < 0.05$]

Table 2 Mean subjective ratings (0–10) of CS+ and CS− slides given at the end of the experiment[a].

	1	2	3	4
CS+	4.4 (2.7)	3.7 (2.5)	2.9 (3.0)	0.2 (0.3)
CS−	0.5 (0.7)	2.0 (2.3)	2.3 (2.1)	4.6 (3.2)
n	11	11	11	12

[a]1, the phobia-relevant CS+/'forward' instruction group; 2, the phobia-relevant CS+/'backward' instruction group; 3, the neutral CS+/'forward' instruction group; 4, the neutral CS+/'backward' instruction group. Standard deviations are given in parentheses. The number of subjects in each group is indicated by n.

reached significance, whereas the interaction of both effects failed to reach significance [$F(1,44) = 1.89$, $p = 0.55$]. These results clearly show that both the stimulus content and the type of instruction had their impact on subjective evaluations of the CS+ and CS−.

*Skin conductance responses**1

A significant main effect of trials occurred during habituation [$F(2,96) = 33.71$, $p < 0.05$], indicating an overall decline in electrodermal responding over trials. As a highly significant instruction × reinforcement interaction [$F(1,48) = 34.85$, $p < 0.05$] made clear, the instructions greatly influenced the response pattern during habituation (left panels, Figure 1): The CS+ evoked higher responses in the 'forward' instruction groups than in the 'backward' instruction groups, with means (√micromho) of 0.90 and 0.52, respectively. The main effect of instruction [$F(1,48) = 4.76$, $p < 0.05$] was largely attributable to this difference. The significant main effect of reinforcement [$F(1,48) = 24.10$, $p < 0.05$] was due to both the impact of the instructions and the fixed order of stimulus presentation in which the CS+ always occurred first. However, the differential responding to CS+ vs CS− slides in these groups disappeared over trials. This was evident from the significant reinforcement × trials interaction [$F(2,96) = 7.99$, $p < 0.05$].

The only main effects that reached significance during acquisition were reinforcement [$F(1,48) = 84.57$, $p < 0.05$], indicating a generally successful conditioning, and trials [$F(5,240) = 11.36$, $p < 0.05$], which was caused by an overall decrease in responding with trials. The means (√micromho) for CS+ and CS− were 0.51 and 0.19, respectively. Except for a significant reinforcement × trials interaction [$F(5,240) = 4.43$, $p < 0.05$] caused by a stronger decrease in responding to CS+ than to CS− slides (middle panels, Figure 1), no significant interactions were found. Since both the stimulus × reinforcement interaction and the stimulus × instruction × reinforcement interaction remained non-significant [for both: $F(1,48) < 1.00$], a better acquisition of the conditioned electrodermal response with phobia-relevant slides could not be demonstrated.

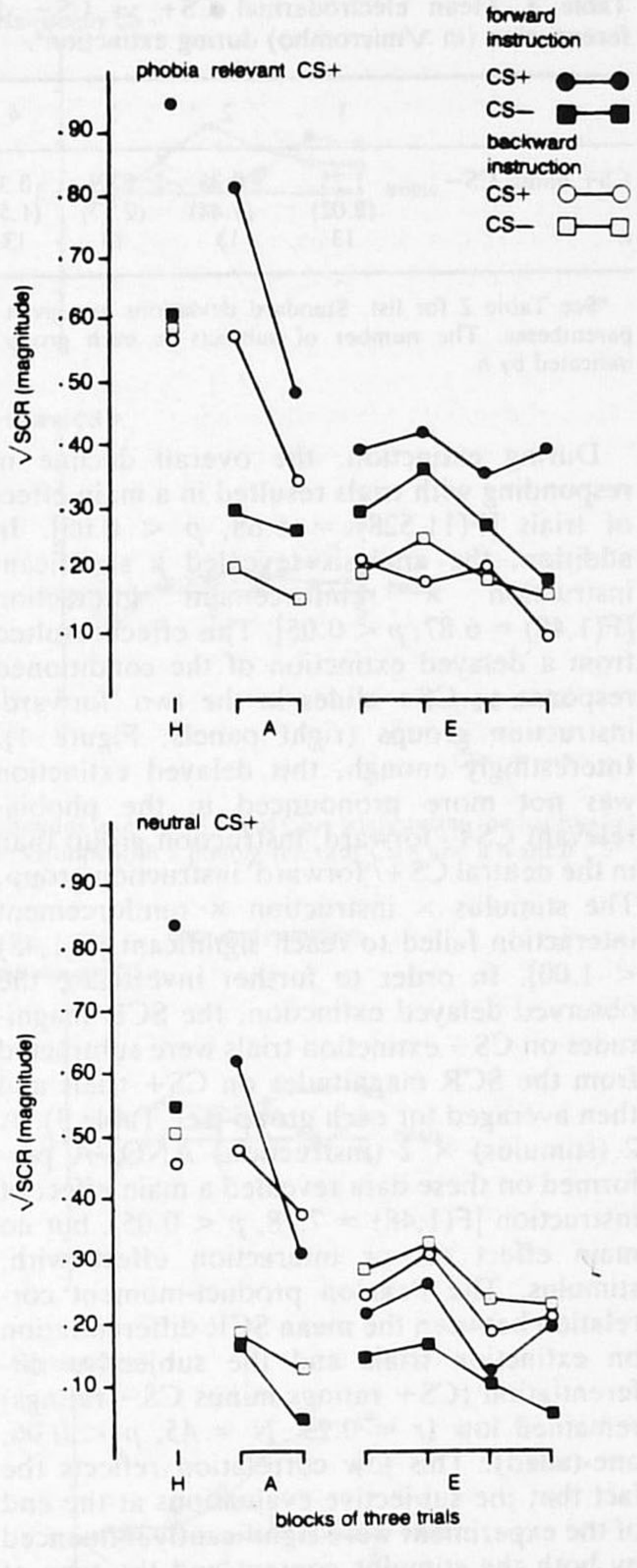


Figure 1 Mean √SCR to reinforced and non-reinforced stimuli (CS+ and CS−, respectively) plotted as a function of trials blocks for (H) habituation, (A) acquisition, and (E) extinction phases of the experiment. Upper row: Groups with a phobia-relevant CS+ and a neutral CS−. Lower row: Groups with a neutral CS+ and a phobia-relevant CS−.

*1 Means and standard deviation on a trial by trial basis of SCRs can be obtained from the first author.

Table 3 Mean electrodermal CS+ vs CS− differentiation (in √micromho) during extinction[a].

	1	2	3	4
CS+ minus CS−	1.25 (2.02)	−0.35 (1.44)	0.89 (2.17)	−0.33 (1.54)
n	13	13	13	13

[a]See Table 2 for list. Standard deviations are given in parentheses. The number of subjects in each group is indicated by n.

During extinction, the overall decline in responding with trials resulted in a main effect of trials [$F(11,528) = 6.68$, $p < 0.05$]. In addition, the analysis revealed a significant instruction × reinforcement interaction [$F(1,48) = 6.87$, $p < 0.05$]. This effect resulted from a delayed extinction of the conditioned response to CS+ slides in the two 'forward' instruction groups (right panels, Figure 1). Interestingly enough, this delayed extinction was not more pronounced in the phobia-relevant CS+/'forward' instruction group than in the neutral CS+/'forward' instruction group. The stimulus × instruction × reinforcement interaction failed to reach significant [$F(1,48) < 1.00$]. In order to further investigate the observed delayed extinction, the SCR magnitudes on CS− extinction trials were subtracted from the SCR magnitudes on CS+ trials and then averaged for each group (see Table 3). A 2 (stimulus) × 2 (instruction) ANOVA performed on these data revealed a main effect of instruction [$F(1,48) = 7.78$, $p < 0.05$], but no main effect of, or interaction effect with, stimulus. The Pearson product-moment correlation between the mean SCR differentiation on extinction trials and the subjective differentiation (CS+ ratings minus CS− ratings) remained low ($r = 0.23$, N = 45, $p < 0.06$, one-tailed). This low correlation reflects the fact that the subjective evaluations at the end of the experiment were significantly influenced by both the stimulus content and the type of instruction, whereas the electrodermal differentiation was mainly affected by the type of instruction.

Heart rate responses

The HRR data of the three experimental phases were subjected to separate stimulus × instruction × reinforcement × trials × beats ANOVAs, with the last three factors being repeated measures. The beats factor consisted of 6 levels (beats 1 through 6, after stimulus onset), and when it reached significance, a follow-up trend-analysis was carried out. Due to equipment failure, the ANOVAs of the three phases were based on different numbers of subjects.

During habituation (n = 47), no significant main effects were found. Irrespective of the instruction, phobia-relevant CS+ evoked an accelerative response pattern (see Figure 2) for the first three beats, as suggested by a significant stimulus × reinforcement × beats interaction [$F(5,215) = 2.70$, $p < 0.05$]. Furthermore, a significant instruction × reinforcement × beats interaction [$F(5,215) = 3.03$, $p < 0.05$] was found. This interaction remained significant when only the first three beats entered into the ANOVA [$F(2,86) = 3.27$, $p = 0.05$], but became non-significant when the ANOVA was restricted to the last two beats [$F(1,43) < 1$]. Thus, it appears that the significant instruction × reinforcement × beats interaction was due to the differential responding to CS+ and CS− slides during the first beats in the groups that received 'forward' instructions.

The ANOVA of the acquisition data (n = 51) revealed a significant effect of beats [$F(5,235) = 3.86$, $p < 0.05$], characterized by a linear trend [$F(1,47) = 6.23$, $p < 0.05$]. As can be seen in Figure 3, an overall accelerative response pattern occurred during the first four beats. The groups with neutral CS+ slides showed, on the whole, a stronger accelerative change from baseline than the groups with phobia-relevant CS+ slides, as suggested by a main effect of stimulus that was close to the significance level [$F(1,47) = 3.95$, $p < 0.06$]. A significant reinforcement × beats interaction [$F(5,235) = 4.59$, $p < 0.05$] indicated that during the last beats the conditioning procedure elicited differential responding to CS+ and CS− trials in all groups. Yet, as a significant stimulus × reinforcement interaction [$F(1,47) = 5.97$, $p < 0.05$] made clear, the pattern of cardiac differentiation was completely different in phobia-relevant CS+ and neutral CS+ groups. When averaged CS+ and CS− heart periods were compared to each other with *a priori* *t*-contrasts, it was found

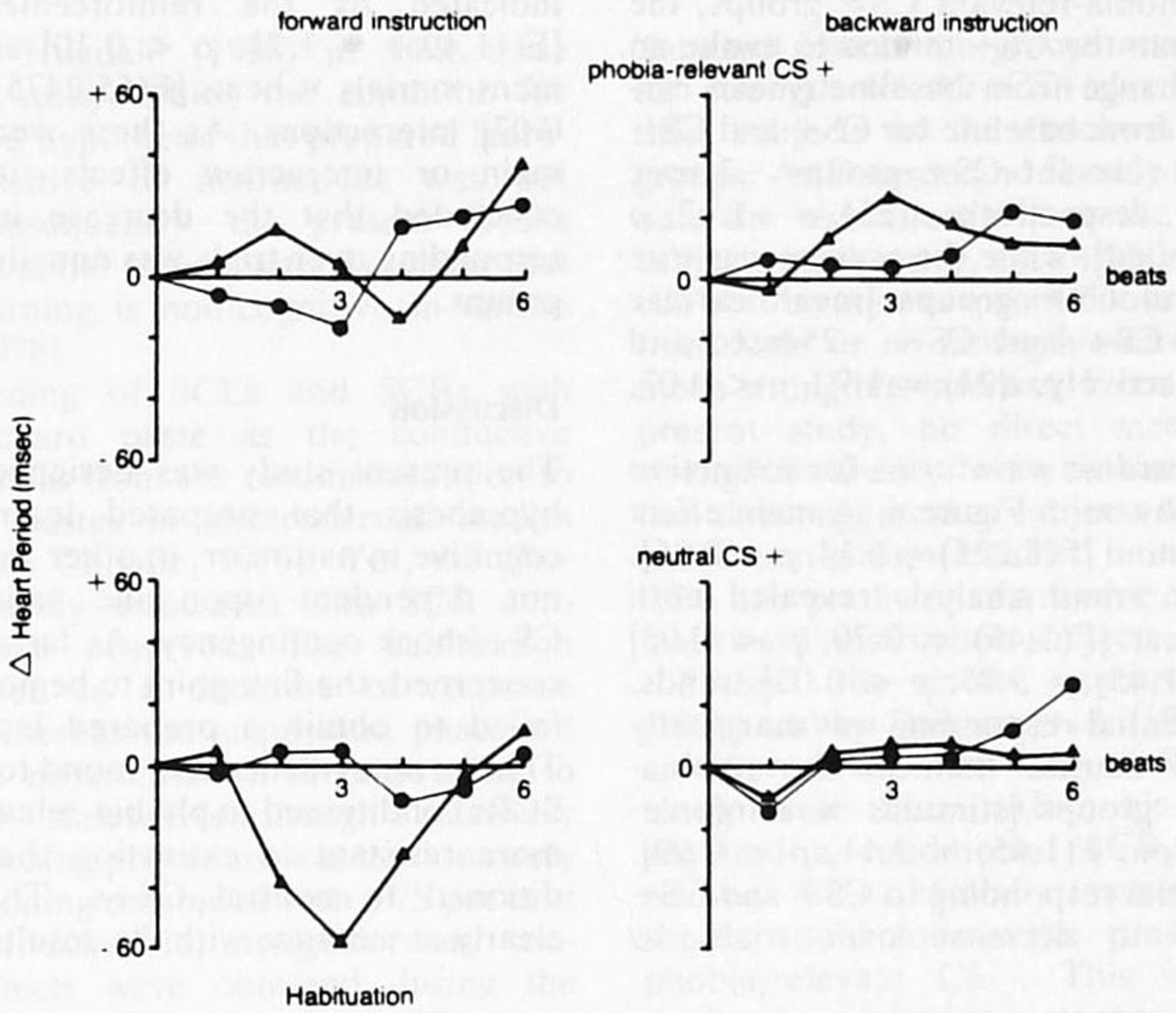


Figure 2 Mean beat-by-beat changes in heart period from pre-stimulus level to CS+ and CS− trials during the habituation phase. Data were collapsed into a single trial block. Upper row: Groups with a phobia-relevant CS+ and a neutral CS−. Lower row: Groups with a neutral CS+ and a phobia-relevant CS−.

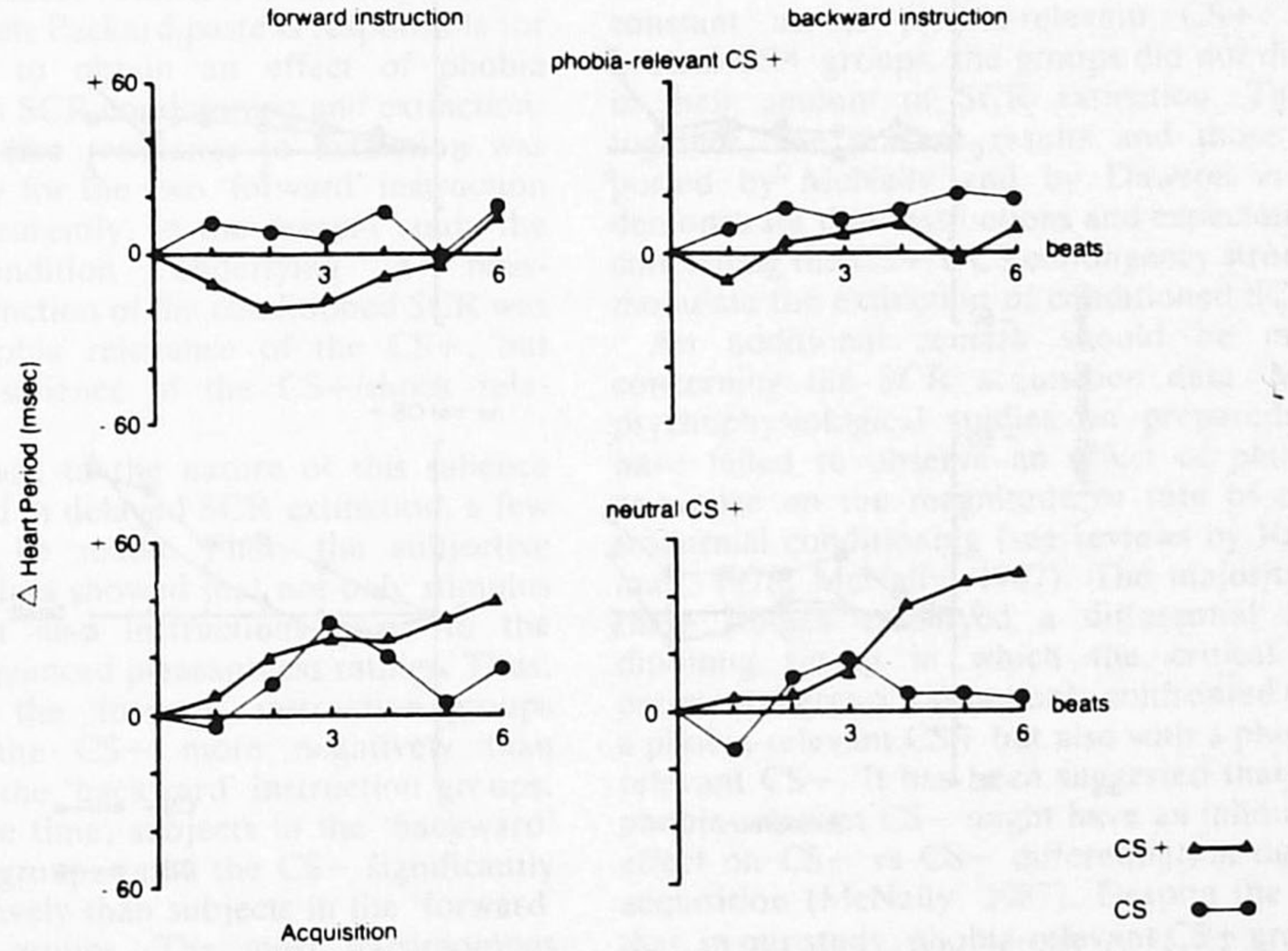


Figure 3 Mean beat-by-beat changes in heart period from pre-stimulus level to CS+ and CS− during the acquisition phase. Data were collapsed into a single trial block. Upper row: Groups with a phobia-relevant CS+ and a neutral CS−. Lower row: Groups with a neutral CS+ and a phobia-relevant CS−.

that, in the phobia-relevant CS+ groups, the CS− rather than the CS+ tended to evoke an accelerative change from baseline [mean cardiac deviation from baseline for CS+ and CS− in the phobia-relevant CS+ groups −3 msec and +11 msec, respectively, $t(25) = -1.62$, $p < 0.12$, two-tailed], while the reverse was true for the neutral CS+ groups [mean cardiac deviation for CS+ and CS− +25 msec and +9 msec, respectively, $t(24) = 1.90$, $p < 0.07$, two-tailed].

The mean cardiac waveforms for extinction (n = 49) are shwon in Figure 4. A main effect of beats was found [$F(5,225) = 6.14$, $p < 0.05$]. The follow-up trend analysis revealed both significant linear [$F(1,45) = 7.70$, $p < 0.05$] and cubic [$F(1,45) = 5.05$, $p < 0.05$] trends. Cardiac differential responding was marginally better in the neutral than in the phobia-relevant CS+ groups [stimulus × reinforcement interaction: $F(1,45) = 3.11$, $p < 0.09$]. Also, differential responding to CS+ and CS− slides tended to decrease over trials, as indicated by the reinforcement × trials [$F(11,495) = 1.71$, $p < 0.10$] and reinforcement × trials × beats [$F(55,2475) = 1.60$, $p < 0.07$] interactions. As there were no further main or interaction effects, it has to be concluded that the decrease in differential responding over trials was equally strong in all groups.

Discussion

The present study was designed to test the hypothesis that prepared learning is non-cognitive in nature or, in other words, that it is not dependent upon the salience of the CS+/shock contingency. As far as the SCR is concerned, the first point to be noted is that we failed to obtain a prepared learning effect. That is, no evidence was found to suggest than SCRs conditioned to phobia-relevent CS+s are more resistant to extinction that SCRs conditioned to neutral CS+s. This finding is clearly at variance with the results obtained by

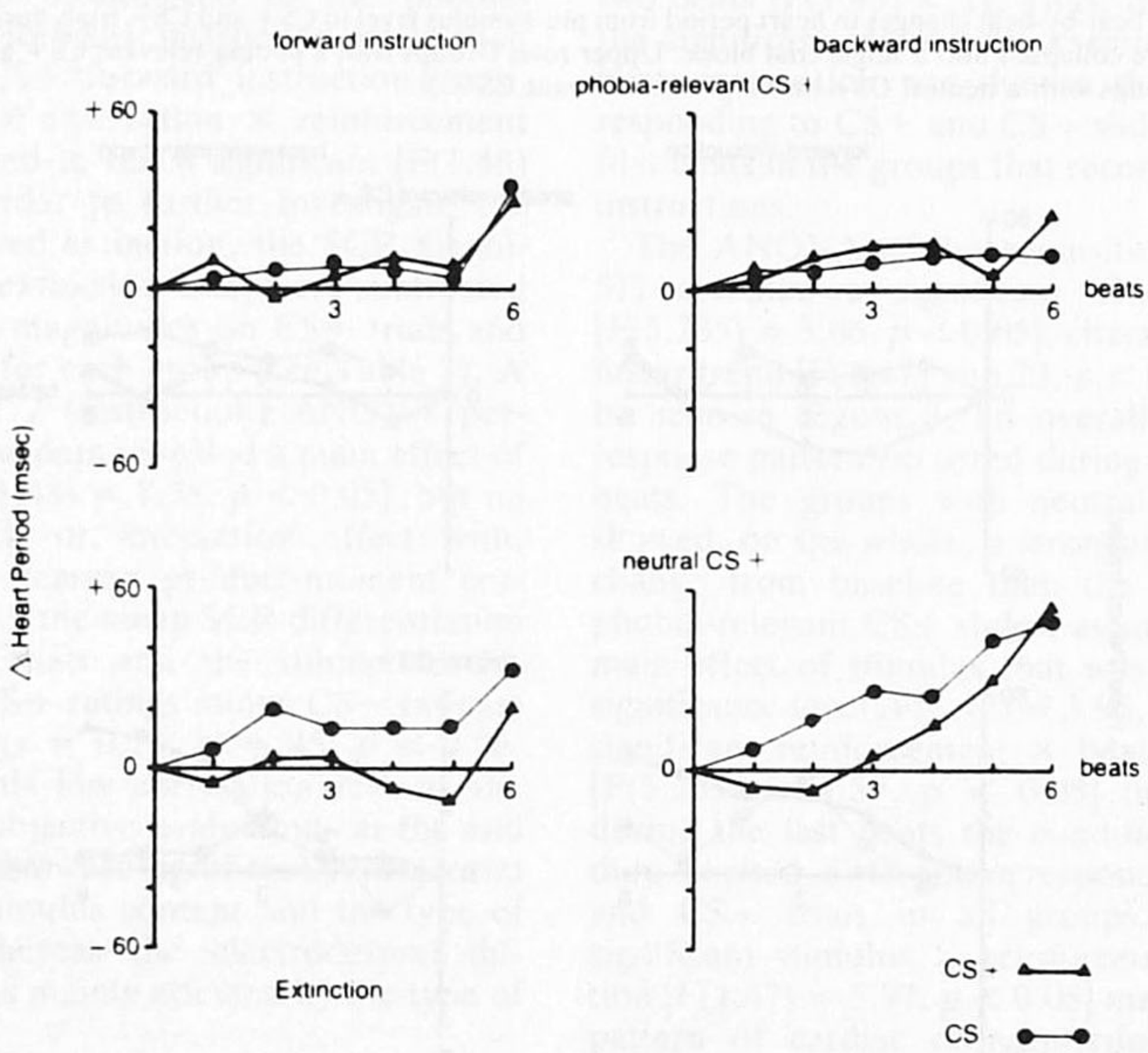


Figure 4 Mean beat-by-beat changes in heart period from pre-stimulus level to CS+ and CS− during the extinction phase. Data were collapsed into a single trial block. Upper row: Groups with a phobia-relevant CS+ and a neutral CS−. Lower row: Groups with a neutral CS+ and a phobia-relevant CS−.

Öhman and Hugdahl (1980) in their trace conditioning study. Thus, the condition for examining the hypothesis that prepared learning is insensitive to instructions was not fulfilled. Consequently, the present results make it impossible to evaluate the claim that prepared learning is non-cognitive in nature (Hugdahl, 1978).

The recording of SCLs and SCRs with Hewlett Packard paste as the conductive medium deviates from the recommendation to use isotonic pastes in electrodermal studies (Venables and Christie, 1973). Yet, the phenomena typically associated with SCR conditioning were observed: The habituation phase resulted in a decrease of response amplitude, whereas the acquisition phase led to a reliable differentiation in responding to CS+ and CS− slides. Even though a relatively weak conditioning procedure was used, namely trace conditioning combined with a 75 per cent reinforcement schedule, straightforward conditioning effects were obtained during the acquisition phase. There were no differences between the groups in this respect. Furthermore, there were no group differences in SCL. Considering these results, it is unlikely that the use of Hewlett Packard paste is responsible for our failure to obtain an effect of phobia relevance on SCR conditioning and extinction. An Öhman-like resistance to extinction was evident only for the two 'forward' instruction groups. Apparently, in the present study the critical condition underlying a resistance to extinction of the conditioned SCR was not the phobia relevance of the CS+, but rather the salience of the CS+/shock relationship.

With regard to the nature of this salience that resulted in delayed SCR extinction, a few points can be made. First, the subjective evaluation data showed that not only stimulus content but also instructions given to the subjects influenced pleasantness ratings. Thus, subjects in the 'forward' instruction groups evaluated the CS+ more negatively than subjects in the 'backward' instruction groups. At the same time, subjects in the 'backward' instruction groups rated the CS− significantly more negatively than subjects in the 'forward' instruction groups. The most parsimonious explanation of these results is that, even after a number of extinction trials, the subjects in the 'forward' instruction groups attributed a greater strength to the CS+/shock contingency than subjects in the 'backward' instruction groups. As Dawson and Furedy (1976) pointed out, the awareness of, or the strength that subjects attribute to, a CS+/shock contingency can best be seen as a continuous scale, ranging from little or no strength/awareness at all to much strength/awareness. Even though, in the present study, no direct measures of this strength/awareness were obtained, the evaluation data suggest that subjects in the 'forward' instruction groups attached more weight to the CS+ as a predictor of aversive events than did 'backward' instruction subjects.

Second, the results reported by McNally (1981) and by Dawson *et al.* (1986) strengthen an interpretation of delayed SCR extinction along these lines. After his subjects had acquired a conditioned SCR to a phobia-relevant CS+, McNally instructed them that shock would follow the presentation of a phobia-relevant CS−. This instruction immediately resulted in a stronger responding to CS− than to CS+ stimuli. Dawson *et al.* (1986) found that when subjective expectancies of shock following CS+ presentations were held constant across phobia-relevant CS+ and neutral CS+ groups, the groups did not differ in their amount of SCR extinction. Taken together, the present results and those reported by McNally and by Dawson *et al.* demonstrate that instructions and expectations concerning the CS+/UCS contingency strongly modulate the extinction of conditioned SCRs.

An additional remark should be made concerning the SCR acquisition data. Most psychophysiological studies on preparedness have failed to observe an effect of phobia-relevance on the magnitude or rate of electrodermal conditioning (see reviews by Rachman, 1978; McNally, 1987). The majority of these studies employed a differential conditioning set-up in which the critical experimental group was not only confronted with a phobia-relevant CS+ but also with a phobia-relevant CS−. It has been suggested that this phobia-relevant CS− might have an inhibitory effect on CS+ vs CS− differentiation during acquisition (McNally, 1987). Despite the fact that, in our study, phobia-relevant CS+ groups saw neutral CS− slides, whereas neutral CS+ groups saw phobia-relevant CS− slides, the

acquisition of conditioned SCRs was as good for neutral CS+ slides as for phobia-relevant CS+ slides. This finding strongly discourages the assumption that the phobia-relevance of CS+s facilitates acquisition of conditioned SCRs.

With regard to the cardiac data, it was found that the conditioning procedure resulted in differential responding to CS+ and CS− trials during acquisition. Unlike the Cook *et al.* (1986) study, we found no difference between phobia-relevant CS+ and neutral CS+ groups as to the absolute amount of cardiac differential responding. Furthermore, whereas Cook *et al.* reported that the conditioned cardiac responses to phobia-relevant CS+ slides were of the accelerative type, we found that cardiac responses on phobia-relevant CS+ trials showed a marked shift from acceleration during habituation to a slight deceleration during acquisition. Thus, while cubic accelerative waveforms have been associated with the 'defensive response' (e.g. Grayson, 1982), we found no evidence of this type of response during acquisition in subjects confronted with phobia-relevant CS+s. This might, of course, be attributable to the difference between the differential delay conditioning paradigm employed by Cook *et al.* and the trace paradigm on which the present study is based. On the other hand, results obtained by Öhman and co-workers suggest that the differential conditioning paradigm, as such, is, in no way, a guarantee that one will observe overall cardiac differential responding, let alone, that one will observe specific accelerative responding to phobia-relevant CS+s (Frederikson and Öhman, 1979; Frederikson, 1981).

It could also be argued that our failure to confirm the Cook *et al.* (1986) findings is related to the fact that the present study used a beat-by-beat format, whereas Cook and colleagues subjected their cardiac data to second-by-second analyses. Admittedly, an analysis in terms of beat-by-beat or heart period has the obvious disadvantage that cardiac activity cannot be associated with stimulus seconds in a straightforward manner (Graham, 1978b). However, most authors agree that, in the end, the choice of either a beat-by-beat or second-by-second format has rather minimal effects on the results (see e.g. Graham, 1978a; Jennings, Berg, Hutcheson, Obrist, Porges, and Turpin, 1981). In view of this contradictory evidence of a phobia-relevance effect on cardiac conditioning, we are reluctant to speculate about the interpretation of the finding that accelerative heart were stronger on neutral CS+ trials than on phobia relevant CS+ trials during the acquisition phase of the present experiment. Interpretations are further complicated by recent papers showing that the associations of cardiac deceleration and acceleration with orienting and defensive responses, respectively, are, to say the least, not yet an established fact (see review by Barry and Maltzman, 1985).

The present study failed to document remaining effects of conditioning on cardiac responses during extinction. In this respect, the present findings confirm the results of previous studies (e.g. Cook *et al.*, 1986; Fredrikson and Öhman, 1979). It is hard to escape the conclusion that HRR is not a promising index to demonstrate resistance to extinction of the conditioned response.

To summarize, in the context of the results reported by earlier studies, the data presented here yield inconclusive evidence of a phobia-relevance effect on cardiovascular conditioning. The electrodermal findings were disappointing in that no effect of phobia-relevance on SCR extinction was obtained. It is, in any event, worth noting that this study is but one of a recent series of electrodermal studies that failed to find evidence of prepared learning (Vaitl, Gruppe, and Kimmel, 1985; McNally and Foa, 1986; Merckelbach, van der Molen, and van den Hout, 1987). While these studies do not rule out the possibility that prepared learning is non-cognitive, they do suggest, however, that prepared learning effects are difficult to obtain and are dependent upon a number of as yet unidentified variables. Further research should focus on the identification of these variables.

More importantly, our SCR data do also indicate that phobia-relevance of the CS+ is certainly not a *sine qua non* for the 'resistance to extinction' phenomenon to occur. This phenomenon can also be observed when the CS+ is neutral, provided the instructions given to the subjects emphasize the CS+/shock relationship. On the assumption that these instructions operate at a cognitive level, the present results point at the importance of cognitive factors in the extinction of conditioned SCRs.

4.7 Fear relevance and diminution of unconditioned skin conductance responses

Summary

According to Donegan and Wagner's priming model (1987), a signalled UCS will elicit a smaller UCR than an unsignalled UCS. Assuming that fear-relevant CSs are good predictors of aversive UCSs, while fear-irrelevant CSs are relatively bad predictors of aversive UCSs, the present study examined the effect of fear relevance on electrodermal UCR diminution during the acquisition phase of a single cue conditioning procedure. Four groups were studied, each of which consisted of 12 subjects. Group 1 had a fear-relevant CS (a slide of an angry face) paired with a UCS (7 mA shock), whereas group 2 had a fear-irrelevant CS (a slide of a happy face) paired with a UCS. Groups 3 and 4 had unpaired presentations of the CS and UCS and served as control groups for groups 1 and 2. There were 4 habituation, 8 acquisition, 4 recovery (UCS-only), and 6 extinction (CS-only) trials. While overall UCR levels were lower in the paired than in the unpaired control groups, it was also found that the size of UCR decrement and subsequent recovery was greater in the angry face CS-paired group than in the happy face CS-paired group. This finding is in line with the predictions that flow from the Donegan and Wagner model.

Key Words: SCR, single cue-conditioning, fear relevance, UCR diminution.

Introduction

Several studies have shown that pairing a neutral, conditioned stimulus (CS) with an aversive, unconditioned stimulus (UCS) does not only result in the appearance of a conditioned response (CR) but also in a CS-linked attenuation of the unconditioned response (UCR) (see reviews by Kimmel, 1966; Dawson & Schell, 1987; Donegan & Wagner, 1987). This latter effect has been termed "UCR diminution", while "UCR recovery" refers to the increase in UCR magnitude which occurs when, after a number of CS-UCS pairings, the UCS is presented without a preceding CS.

Electrodermal (Grings & Schell, 1971), cardiovascular (Lykken, Macindoe & Tellegen, 1972; exp. 1), evoked potential (Lykken et al., 1972; exp. 2), and eyeblink (Kimble & Ost, 1961) studies have reported reliable UCR diminution and recovery effects. Furthermore, parametric studies have shown that variables that are known to affect the CS in aversive Pavlovian conditioning are also of relevance to UCR diminution and recovery. Thus, the number of reinforced trials correlates positively with the amount of UCR diminution and subsequent recovery (Kimmel & Pennypacker, 1962), the amount of diminution and recovery vary with interstimulus intervals in exactly the same manner as CS conditioning does (Kimble & Ost, 1961; Grings & Schell, 1971), and, finally, the presentation of CS-only trials leads to an extinction of UCR diminution (Morrow, 1966). An interpretation of these effects in terms of effector fatigue or CR/UCR interference (Grings & Schell, 1969) is not convincing, as it is a well-established fact that UCR diminution and recovery can also be found in trace conditioning set-ups, i.e., in conditioning procedures in which there is a time gap between CS offset and UCS onset (Grings & Schell, 1971). All in all, there can be no doubt that UCR diminution and recovery are genuine conditioning phenomena, with UCR diminution reflecting a conditioned inhibition controlled by the CS and UCR recovery reflecting a release of this inhibition by CS omission (Lykken & Tellegen, 1974).

With regard to the theoretical interpretation of these phenomena, Donegan and Wagner (1987) proposed a "priming" model which is based on an information processing approach to conditioning. The Donegan and Wagner model rests on two basic assumptions. The first assumption is that "primed", i.e., signalled, events are less effectively processed in short-term memory than unsignalled events. The von Restorff effect can be taken as empirical support for this assumption (van den Hout, Zijlstra & Merckelbach, 1988). A second, more theoretical, assumption is that the magnitude of the UCR indexes the depth of processing of the UCS in short-term memory. The implication of these assumptions is that a CS-signalled UCS will be less effectively processed in short-term memory and, therefore, less successful in eliciting a UCR than an unsignalled UCS.

Using electric shock as the UCS and fear-relevant stimuli (e.g., slides of snakes, fearful or angry faces) or fear-irrelevant/neutral

stimuli (e.g., slides of mushrooms or happy faces) as CSs, various studies have found that electrodermal responses, once conditioned, extinguish more slowly to fear-relevant than to fear-irrelevant/neutral CSs (see reviews by Öhman, 1986; Dimberg, 1986; McNally, 1987), thereby confirming a number of predictions that can be derived from Seligman's "preparedness" hypothesis (1971). However, virtually no attention has been paid to the effect of the fear relevance of the CS on the development of the UCR during aversive conditioning. In what seems to be the only study with data bearing on this topic, Orr and Lanzetta (1980) reported a decrease in electrodermal UCRs for subjects who were shown a slide of a fearful face as the CS and an increase for subjects shown a happy face CS. One explanation for Orr and Lanzetta's finding might be that fear relevance is associated with an increased degree of UCR diminution. More specifically, following Donegan and Wagner's model (1987), one would expect an evolutionary "belongingness" between a CS and an aversive UCS to result in the CS being a better predictor of the UCS, which, in turn, would result in less effective UCS processing and greater UCR diminution. Similarly, one would expect a CS that is incompatible with an aversive UCS (e.g., a happy face) to be associated with less UCR diminution. Assuming that such a CS is a bad predictor of aversive UCSs, CS-UCS pairings should result in a high level of UCS processing and, consequently, in a reduced UCR diminution.

The present study was carried out in order to explore the relationship between fear relevance and UCR diminution. Drawing on Donegan and Wagner's model, the hypothesis tested was that fear-relevant CSs will induce more UCR diminution and recovery than fear-irrelevant CSs. Using a simple conditioning procedure, the electrodermal UCR diminution in subjects given a fear-relevant CS was compared to the UCR diminution in subjects given a fear-irrelevant CS.

Method

Subjects

The subjects were 48 students (22 males and 26 females), all of whom received some small financial compensation for their participation

in the experiment. Their mean age was 21.1 years (range: 18-33 years).

Apparatus and Stimulus Materials

Skin conductance level (SCL) and skin conductance response (SCR) were picked up using two Beckman Ag-AgCL electrodes (8 mm diameter) attached to the medial phalanges of the second and third fingers of the subject's right hand and connected to a Beckman Skin Conductance Coupler (type 9844). SCL and SCR recordings were based on the constant voltage (.5 V) method. The coupler allowed for a maximum sensitivity of 0.05 micromho.

Respiration was recorded using a Beckman Respiration Belt, fastened around the subject's chest. The respiration belt was connected to a Beckman Pressure/Pulse/Voltage Coupler (type 9884). SCR and respiration were monitored on a Beckman R 711 polygraph.

CSs were slides of angry and happy faces, taken from Ekman and Friesen (1975). A Kodak Carousel was used for presentation of the slides, which were projected onto a white screen. The size of the projected image was approximately 75 x 110 cm, and it appeared 2 m in front of the subject.

A capacitor was used for administration of the shock UCSs. The shocks were delivered through two electrodes attached to the first finger of the subject's left hand. The shock intensity was set at 7 mA (dc).

Slide presentation, shock administration, and intertrial intervals were controlled by a PDP Minc-11 microcomputer.

Design

A 2 x 2 between-subject design was used, with type of CS (angry face CS vs. happy face CS) as the first between-subject factor and conditioning (paired vs. unpaired CS-UCS presentations) as the second between-subject factor. Thus, four groups were studied, each of which consisted of 12 subjects. In group 1, an angry face CS was paired with shock, whereas in group 2, a happy face CS was paired with shock. Groups 3 (angry face) and 4 (happy face) served as control groups in which the

CS and shocks were explicitly unpaired. Truly random control groups were not used, as it has been shown that explicitly unpaired CS-UCS presentations provide an appropriate control procedure for simple conditioning (Furedy & Schiffman, 1971).

Procedure

Each subjects was seated in a comfortable chair placed in a dimly lit, sound-attenuated chamber. Recording apparatus and the Kodak Carousel were located in an adjacent room. Slides were projected through a hole in the wall. After the subject had filled in the fear questionnaire (FQ; Marks & Mathews, 1979), electrodermal recording sites were cleaned with distilled water and the electrodes and respiration belt were attached. The experiment was described to the subject as a study of physiological reactions to different sensory stimuli. No information about the contingency between CS and UCS was given. Next, the subject received a series of shocks of increasing intensity until the 7 mA level was reached. He or she was then asked to indicate on a 14 cm visual analog scale, with 0 indicating "not painful at all" and 14 indicating "extremely painful", how painful he or she thought the 7 mA shock was.

A single-cue conditioning procedure was then carried out with each subject seeing only one CS slide. The subject was first given 4 CS-only (habituation) trials. An acquisition phase followed in which groups 1 and 2 received 8 CS-UCS pairings. Shock UCSs were delivered exactly at CS offset. For groups 3 and 4, CS and UCS were presented unpaired. That is, one stimulus was presented at the beginning of the intertrial interval and the other stimulus was presented at the midpoint of this interval. A recovery phase consisting of 4 UCS-only presentations followed. Finally, 6 extinction (CS-only) trials were given.

CSs had a duration of 5 sec and shocks had a duration of 0.5 sec. Intertrial intervals varied from 20 to 40 sec and had a mean of 30 sec. In order to control for irrelevant aspects of the CSs, different subjects within the same group saw different CSs.

Response Definition and Analysis

SCLs were measured on four occasions: Before habituation, between habituation and acquisition, between recovery and extinction, and at the end of the extinction phase. SCRs were defined as the maximal deflections occurring 1-5 sec after CS or UCS onset. SCL and SCR were measured in micromho and square root-transformed.

Respiration was used as control variable. Following the criteria of Stern, Ray, and Davis (1980), trials with respiratory irregularities were excluded from statistical analyses. Such irregularities occurred on less than 2% of the trials. SCR values for these trials were estimated on the basis of the SCRs on adjacent trials. As UCR diminution and recovery are generally seen as phenomena associated with classical conditioning, it is obligatory to ascertain that conditioning actually occurred in groups receiving paired CS-UCS presentations. Consequently, not only UCRs, but also CRs were examined in the present study.

Not only were absolute UCR levels taken into account, but also, and most importantly, the size of UCR decrement and recovery. Thus, for each subject, a UCR diminution score and a UCR recovery score were calculated (Kimmel & Pennypacker, 1962). The former score consisted of the average SCR to the first two shocks during acquisition minus the average SCR to the last two shocks during acquisition. The latter score consisted of the mean SCR on the recovery trials minus the mean SCR to the last two shocks during acquisition. Greater diminution values reflect a larger UCR decrement, whereas positive recovery values indicate that the mean UCR on recovery trials is greater than that on the final acquisition trials.

FQ scores, subjective evaluation of the shock, SCLs, SCRs to CS and UCS, and UCR diminution and recovery scores were subjected to 2 (fear relevance: angry face CS vs. happy face CS) x 2 (conditioning: paired CS-UCS vs. unpaired CS-UCS presentations) analyses of variance (ANOVAs).

For ANOVAs involving a trial factor as the repeated measure, Greenhouse-Geisser corrections were used. The trial factor consisted of blocks of two trials.

Results

FQ, Subjective Evaluation of the UCS, and SCLs

Table 1 shows the mean FQ scores as well as the mean subjective evaluations of the 7 mA shock UCS of the four groups. The 2 (fear relevance) x 2 (conditioning) ANOVAs revealed no significant group differences for either FQ scores or evaluation of the UCS.

SCL data were subjected to a 2 (fear relevance) x 2 (conditioning) x 4 (occasions) ANOVA, with the last factor having repeated measures. Again, no significant group differences emerged. The main effect of occasions was the only source reaching significance [$F(3,132)=14.1$, $p<0.05$], due to a systematic SCL increase during the experiment in all groups.

Table 1. MEAN FQ SCORES AND UCS EVALUATIONS (0-14) OF THE FOUR GROUPS (STANDARD DEVIATIONS ARE GIVEN IN PARENTHESES).

Groups*	1	2	3	4
FQ	21.5 (11.8)	22.5 (14.5)	20.8 (9.5)	22.8 (14.1)
UCS evaluation	4.6 (1.9)	4.8 (1.6)	4.6 (1.6)	5.5 (1.2)

* 1; the angry face CS-paired group, 2; the happy face CS-paired group, 3; the angry face CS-unpaired group, 4; the happy face CS-unpaired group.

SCRs to CSs

SCRs during habituation trials (left panel, Figure 1) were evaluated with a 2 (fear relevance) x 2 (conditioning) x trials (blocks of two trials) ANOVA, with the last factor having repeated measures. Except for a significant main effect of trials [$F(1,44)=17.3$, $p<0.05$], which was caused by a decrease in SCRs over trials, no further significant main or interaction effects were found.

Analysis of the acquisition data (middle panel, Figure 1) revealed a main effect of conditioning [$F(1,44)=4.3$, $p<0.05$], caused by the overall

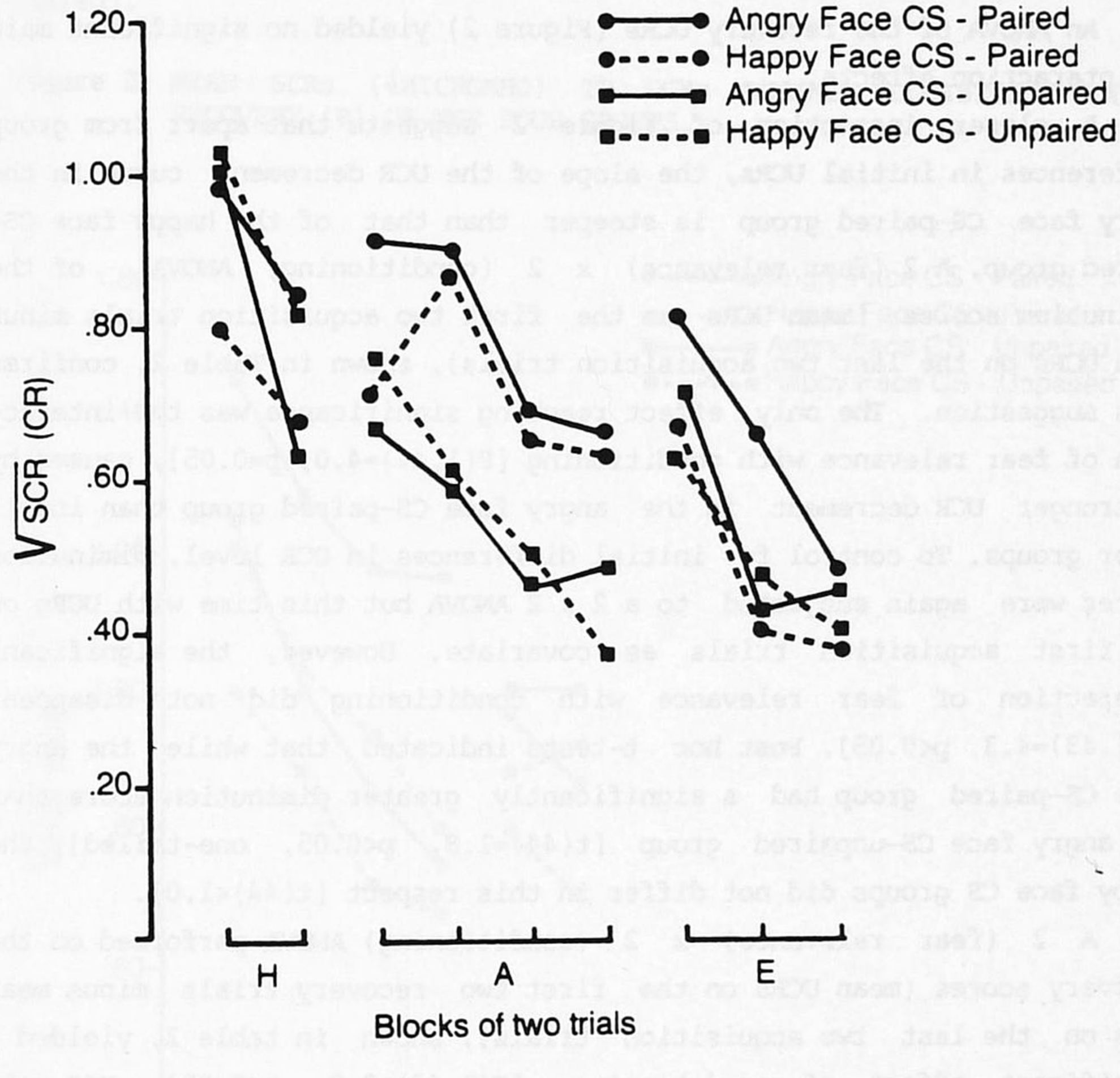
higher level of responding to the CS in groups with paired CS-UCS presentations than in groups with unpaired presentations. Furthermore, a significant main effect of trials (blocks of two trials) emerged [$F(3,132)=14.6$, $p<0.05$]. This effect was due to a general decline in responding over acquisition trials. Conditioning effects in the angry face CS-paired group were not stronger than those in the happy face CS-paired group; both the fear relevance x conditioning interaction [$F(1,44)<1.0$] and the fear relevance x conditioning x trials interaction [$F(3,132)=1.2$, $p=0.31$] remained non-significant.

As regards the extinction data (right panel, Figure 1), the overall decrease in responding resulted in a main effect of trials (blocks of two trials) [$F(2,88)=25.4$, $p<0.05$]. The main effect of conditioning remained non-significant [$F(1,44)<1.0$]. This can be attributed to the fact that responding in the happy face CS-paired group did not differ from responding in the unpaired groups. Though not significant, the fear relevance x conditioning x trials interaction [$F(2,88)=1.7$, $p=0.20$] suggested a resistance to extinction in the angry face CS-paired group. To further examine this effect, separate **post hoc** t-tests were carried out for each extinction trial block. Only for the second extinction block, the difference in CRs between the angry face CS-paired group and all other groups approached significance [$t(44)=1.6$, $p=0.06$, one-tailed].

SCRs to UCSs

A 2 (fear relevance) x 2 (conditioning) x trials (blocks of two trials) ANOVA, performed on the SCRs to shocks during acquisition (Figure 2), revealed a significant main effect of conditioning [$F(1,44)=5.3$, $p<0.05$]: Overall, UCRs in the paired groups were smaller than those in the unpaired groups. In addition, a borderline significant effect of fear relevance was found [$F(1,44)=3.6$, $p=0.06$], due to the larger UCRs in the angry face CS-unpaired and angry face CS-paired groups than in the happy face CS-unpaired and happy face CS-paired groups, respectively. The significant main effect of trials [$F(3,132)=44.1$, $p<0.05$] was caused by a general decrease in responding over trials.

Figure 1. MEAN SCRs (√MICROMHO) TO CSs DURING HABITUATION (H), ACQUISITION (A), AND EXTINCTION (E) OF THE FOUR GROUPS.



As a fear relevance x conditioning x trials interaction suggested, UCR diminution tended to be weaker in the angry face CS-unpaired group than in all other groups [$F(3,132)= 1.9$, $p=0.14$].

Examination of UCRs on the first acquisition trial revealed a main effect of fear relevance [$F(1,44)=4.2$, $p<0.05$]: Angry face CS groups had significantly larger initial UCRs than the happy face CS groups, the means (√micromho) being 1.46 (s.d.=0.50) and 1.18 (s.d.=0.44), respectively.

An ANOVA of the recovery UCRs (Figure 2) yielded no significant main or interaction effects.

A closer inspection of Figure 2 suggests that apart from group differences in initial UCRs, the slope of the UCR decrement curve in the angry face CS-paired group is steeper than that of the happy face CS-paired group. A 2 (fear relevance) x 2 (conditioning) ANOVA of the diminution scores (mean UCRs on the first two acquisition trials minus mean UCRs on the last two acquisition trials), shown in Table 2, confirms this suggestion. The only effect reaching significance was the interaction of fear relevance with conditioning [$F(1,44)=4.0$, $p=0.05$], caused by a stronger UCR decrement in the angry face CS-paired group than in all other groups. To control for initial differences in UCR level, diminution scores were again subjected to a 2 x 2 ANOVA but this time with UCRs on the first acquisition trials as covariate. However, the significant interaction of fear relevance with conditioning did not disappear [$F(1,43)=4.3$, $p<0.05$]. **Post hoc** t-tests indicated that while the angry face CS-paired group had a significantly greater diminution score than the angry face CS-unpaired group [$t(44)=1.8$, $p<0.05$, one-tailed], the happy face CS groups did not differ in this respect [$t(44)<1.0$].

A 2 (fear relevance) x 2 (conditioning) ANOVA performed on the recovery scores (mean UCRs on the first two recovery trials minus mean UCRs on the last two acquisition trials), shown in table 2, yielded a significant effect of conditioning [$F(1,44)=3.9$, $p<0.05$]: UCS-only trials elicited a stronger UCR increase in paired groups than in unpaired groups. As a marginally significant interaction of fear relevance with conditioning [$F(1,44)=2.4$, $p=.13$] suggested, the UCR increase tended to be stronger in the angry face CS-paired group.

Post hoc t-tests showed, indeed, that the difference in UCR recovery between the angry face CS-paired group and the unpaired groups [$t(44)=2.1$, $p<0.05$, one-tailed] was greater than that between the happy face CS-paired group and the unpaired groups [$t(44)=1.2$, $p=0.14$, one-tailed]. Furthermore, whereas the happy face CS groups had comparable recovery scores [$t(44)<1.0$], the angry face CS-paired group showed a stronger recovery than the angry face CS-unpaired group [$t(44)=2.5$, $p<0.05$, one-tailed].

Figure 2. MEAN SCRs ($\sqrt{}$MICROMHO) TO UCRs DURING ACQUISITION (A) AND RECOVERY (R) OF THE FOUR GROUPS.

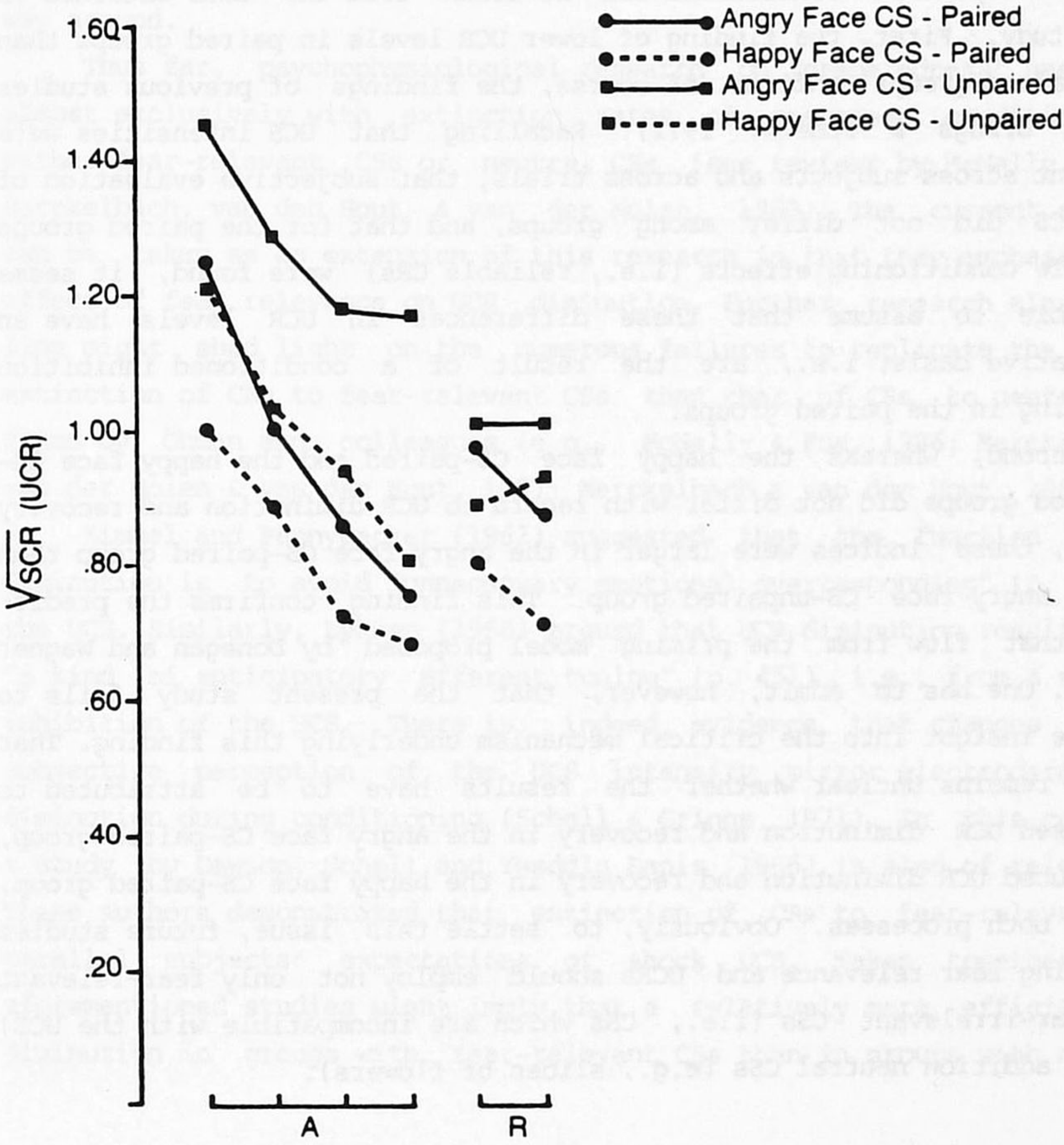


Table 2. MEAN DIMINUTION AND RECOVERY SCORES (√MICROMHO) OF THE FOUR GROUPS (STANDARD DEVIATIONS ARE GIVEN IN PARENTHESES).

Groups*	1	2	3	4
Diminution	0.51 (0.37)	0.32 (0.27)	0.28 (0.24)	0.44 (0.30)
Recovery	+0.22 (0.32)	+0.13 (0.26)	−0.05 (0.20)	+0.09 (0.29)

*See Table 1 for list.

Discussion

Two important conclusions can be drawn from the data obtained in this study. First, the finding of lower UCR levels in paired groups than in unpaired groups confirms, of course, the findings of previous studies (e.g., Grings & Schell, 1971). Recalling that UCS intensities were constant across subjects and across trials, that subjective evaluation of the UCS did not differ among groups, and that for the paired groups reliable conditioning effects (i.e., reliable CRs) were found, it seems plausible to assume that these differences in UCR levels have an associative basis, i.e., are the result of a conditioned inhibition operating in the paired groups.

Second, whereas the happy face CS-paired and the happy face CS-unpaired groups did not differ with regard to UCR diminution and recovery scores, these indices were larger in the angry face CS-paired group than in the angry face CS-unpaired group. This finding confirms the predictions that flow from the priming model proposed by Donegan and Wagner (1987). One has to admit, however, that the present study fails to provide insight into the critical mechanism underlying this finding. That is, it remains unclear whether the results have to be attributed to increased UCR diminution and recovery in the angry face CS-paired group, to reduced UCR diminution and recovery in the happy face CS-paired group, or to both processes. Obviously, to settle this issue, future studies regarding fear relevance and UCRs should employ not only fear-relevant and fear-irrelevant CSs (i.e., CSs which are incompatible with the UCS) but in addition neutral CSs (e.g., slides of flowers).

Interpretation of the UCR diminution and recovery data is also complicated by differences in initial UCRs: Regardless of whether or not the CS and UCS were paired, groups with an angry face CS had higher initial UCRs than groups with a happy face CS. On the one hand, the facts that UCS intensities were kept constant across groups and that subjective estimates of the UCS did not differ among groups seem, at first, to suggest that initial UCR differences were due to chance. On the other hand, it may well be that confrontation with a fear-relevant CS potentiated responding to the UCS. Öhman, Eriksson, Fredrikson, Hugdahl, and Olofsson (1974; exp. 4) reported that the administration of shocks prior to habituation increases differential responding to fear-relevant and neutral CSs during habituation. Yet, there is no **a priori** reason to exclude the possibility that such a process might also operate the other way around.

Thus far, psychophysiological research on "preparedness" has dealt almost exclusively with extinction rates of responses conditioned to either fear-relevant CSs or neutral CSs (see reviews by McNally, 1987; Merckelbach, van den Hout & van der Molen, 1989). The current results can be taken as an extension of this research in that they emphasize the effect of fear relevance on UCR diminution. Further research along this line might shed light on the numerous failures to replicate the slower extinction of CRs to fear-relevant CSs than that of CRs to neutral CSs found by Öhman and colleagues (e.g., McNally & Foa, 1986; Merckelbach, van der Molen & van den Hout, 1987; Merckelbach & van den Hout, 1988).

Kimmel and Pennypacker (1962) suggested that the function of UCR diminution is to avoid "unnecessary emotional overresponding" (p. 23) to the UCS. Similarly, Lykken (1968) argued that UCR diminution results from "a kind of anticipatory afferent tuning" (p. 451), i.e., from a sensory inhibition of the UCS. There is, indeed, evidence that changes in the subjective perception of the UCS intensity mirror electrodermal UCR diminution during conditioning (Schell & Grings, 1971). In this context, a study by Dawson, Schell and Tweddle Banis (1986) is also of relevance. These authors demonstrated that extinction of CRs to fear-relevant CSs parallel subjects' expectations of shock UCS. Taken together, the aforementioned studies might imply that a relatively more efficient UCR diminution in groups with fear-relevant CSs than in groups with neutral

or fear-irrelevant CSs results in the former groups attributing less significance to the UCS. In the long run, this would offset any initial differences in CRs and UCRs between fear-relevant CS and fear-irrelevant or neutral CS groups. The net effect of this hypothesized process would be equal CR extinction rates in fear-relevant and fear-irrelevant CS groups. This might especially be true for studies in which high intensity UCSs are employed, since there is reason to assume that high intensity UCSs promote a more efficient UCR diminution (Donegan & Wagner, 1987).

As regards the CRs found in the present study, the absence of a facilitating effect of fear relevance on CR development during acquisition is in line with the earlier research of Dimberg (1986; 1987). This author concluded that the critical effect of fear relevance on CRs manifests itself during extinction. Although the present study found some evidence for a resistance to extinction of SCRs conditioned to angry faces, this effect was not as strong as that reported by Dimberg. Yet, it should be noted that UCS-only trials (i.e., recovery trials) preceded the extinction phase. It is a well established fact that such UCS-only trials elicit post-conditioning re-evaluation of the UCS and, consequently, undermine the occurrence of CRs during subsequent extinction (Davey, 1987).

4.8 Summary

The present author's contribution to the empirical work in the field of preparedness can be summarized as follows:

1. There is evidence which suggests that the hypothesized prepared fear of animals such as rats, snakes, and spiders is related to a set of salient characteristics (e.g., deviant appearance, strange tactile impression, unpredictability) of these animals (section 4.1). This reminds us of Hull's (1943) assertion that the effectivity of a CS during conditioning partly depends on CS characteristics (e.g., dynamism, intensity, salience)[1]. Hence, the question arises whether the delayed extinction of responses conditioned to the so-called phobia-relevant CSs found by Öhman (1986) is a function of the survival value or of the salience of these CSs (see section 6.4).
2. An issue closely related to this question was discussed in section 4.2. The results presented in that section indicate that phobia-relevant CSs differ more from neutral CSs in terms of the dangerousness and unpredictability that subjects ascribe to these CSs than in terms of survival relevance (as rated by experts).

   The studies presented in the first two sections can best be looked upon as initial attempts to develop an alternative to the survival relevance or preparedness interpretation of the selectivity of fears. The third and fourth studies sought to examine the credibility of the preparedness interpretation in more direct ways.
3. Thus, in the third study (section 4.3), fears in a severe, clinical sample, were rated for survival relevance. Contrary to the results of earlier studies (e.g., De Silva et al., 1972), it was found that there was no preponderance of survival-relevant fears in this sample. Additionally, it became clear that the fears that meet the criteria for being classified as relatively survival-relevant are associated with a poor treatment outcome. Unfortunately, this positive finding was articulated in section 4.3. Recalling Seligman's claim (1971; De

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[1] Referring to Hull (1934), McAllister and McAllister (1979) rightly remarked that Seligman's (1971) qualification of the older conditioning theories as being wholly based on the premise of equipotentiality is an overstatement of the case.

Silva et al., 1977) that most phobic and obsessive fears pertain to survival-relevant stimuli, a discussion that emphasizes the failure to support this claim would have been more to the point (see section 6.2).

4. Öhman et al. (1985) proposed that not only animal phobias but social phobias, too, are the result of prepared conditioning. According to their analysis (section 2.3), facial cues play a pivotal role in this conditioning. However, the study presented in section 4.4 did not find any evidence for the notion that social phobics react more strongly to facial cues than normal control subjects.

   The last three psychophysiological studies consisted of conditioning experiments that were carried out in order to examine some crucial aspects of the conditioning phenomena documented by Öhman (1986).

5. The fifth study (section 4.5) did not succeed in observing a delayed extinction of SCRs conditioned to phobia-relevant stimuli (i.e., snakes, spiders). This result is at variance with Öhman's (1979) claim that conditioned responses directed at prepared cues are resistant to extinction (section 3.4).

6. In the sixth study (section 4.6), it was demonstrated that SCRs conditioned to phobia-relevant (i.e., snakes, spiders) and neutral (i.e., mushrooms, flowers) CSs are equally prone to verbal manipulation. It was found that instructions stressing the CS+\UCS contingency elicited a delayed extinction of conditioned SCRs both in subjects looking at phobia-relevant CSs and subjects looking at neutral CSs. Additionally, the results indicated that instructions masking the CS+\UCS contingency not only enhanced the extinction of SCRs conditioned to neutral CS+s but also the extinction of SCRs conditioned to phobia-relevant CS+s. This finding is difficult to reconcile with the idea that SCRs directed at prepared cues are of a non-cognitive nature (section 3.4).

7. The final study examined an issue that has received little or no attention in psychophysiological experiments concerned with prepared conditioning. This study attempted to find out whether CS content does or does not affect the development of conditioned UCR diminution. Evidence suggestive of a more strongly conditioned UCR diminution with phobia-relevant (i.e., angry faces) than with control (i.e.,

happy faces) stimuli was found. This finding is important because it may imply that the phobia-relevant stimuli employed by Öhman are associated both with a delayed extinction of conditioned SCRs and with an enhanced UCR diminution (see section 7.2).

CHAPTER FIVE

A THEORETICAL CRITIQUE

From whichever angle one prefers to look at the preparedness hypothesis, its central message is that phobic behavior is rooted in evolutionary processes. "Wilson's ladder" is unmistakably present in both Seligman's and Öhman's version of the preparedness hypothesis. The aim of the present chapter is to show that, as far as the preparedness concept is concerned, this ladder has some serious defects. In the first section, Seligman's position is critically evaluated. The second section takes issue on Öhman's distinction between open and closed genetic programs. The third section deals with Öhman's theory about the evolutionary origins of animal and social phobias.

5.1 The danger of analogies

It is not stretching the point too far to summarize Seligman's (1971) argumentation as follows: Both phobias in humans and taste aversion in animals belong to a greater family of behaviors that serve an adaptive function. Thus, phobias are similar to taste aversion and, as a result, must possess the characteristics of taste aversion.

Bold analogies are certainly not uncommon in analyses of human behavior inspired by evolutionary theories (Benedict, 1979). To quote just one example,

> "Boobies sky-point with their beaks, the Masai thrusts his spear into the ground, and in our culture we salute state visitors with twenty-one averted guns. Similarities of this kind call for an explanation. They can be accidental, but most of the time they are not, being due rather to similar selective pressures" (Eibl-Eibesfeld, 1979; p. 2).

There is nothing wrong with the use of analogies in the initial stage of theory building. On the contrary, as the famous example of Kékulé's dream of circling snakes and his subsequent discovery of the

structure of the benzeen molecule illustrates, analogies can be extremely fruitful. However, doubt arises at the point were they are used both as tools that generate hypotheses and as sources that may provide empirical support for these hypotheses. It is exactly this confusing role that the taste aversion analogy has in Seligman's (1971) version of the preparedness hypothesis. For instance, Seligman (1971) propounds that phobias are a form of prepared learning because they have the defining characteristic of being acquired quickly, then remarks "let us now look at the evidence that leads in this direction" (p. 314) and goes on to quote examples from the taste aversion literature which should serve to show that phobias are, in addition, resistant to extinction, non-cognitive etc..

As the quotation in the introduction to chapter three made clear, Seligman and co-workers recognized the glibness of evolutionary arguments. Yet, this same quotation is revealing in that it shows how strongly Seligman's ideas deviate from hypotheses commonly held in evolutionary biology. Although it is hardly fit for serious discussion, let us, just for the sake of the argument, pursue Seligman's research proposal. Let us assume that there is an anthropologist who discovers a primitive tribe that functions at a prehistoric level. Let us further assume that a longitudinal community study of this tribe succeeds in documenting that the prevalence of fear of the dark increases whereas the prevalence of avoidance of eating in public decreases through the ages. The assertion that this finding would testify to the evolutionary origins of phobias is questionable in at least two respects. In the first place, unless it can be demonstrated that the two types of phobias are under differential genetic control, such an assertion seriously runs the risk of becoming a Lamarckian and, therefore, a scientifically untenable speculation. The argument that people may transmit their adaptive fears to the next generation by non-genetical means can, of course, be advanced. But this is just to say that we do not need evolutionary and genetical constructs to account for the selectivity of fears.

In the second place, even if it could be shown that phobias are under differential genetic control, the above assertion is still dubious. It is based on the incorrect view that natural selection acts on single characteristics. In some rare instances in which one trait is known to affect survival to a great extent this view might be justified. Kettle-

well (1973), for example, was able to show that natural selection acts on the coloration of the peppered moth to the effect that the frequency of a black coloration increases in polluted, urban environments while the speckled form predominates in rural environments. He demonstrated, in a straightforward manner, that in polluted environments with black tree-trunks a black coloration confers superior camouflage and, consequently, diminished risk of predation. Yet, in the case of prehistoric man, it is not unrealistic to assume that survival did depend upon many more characteristics than just fear and avoidance of dangerous situations. This means that in his case, selection pressures acted on the total organism rather than on an isolated characteristic (Hailman, 1979). It could be countered that under **ceteris paribus** conditions the assertion formulated above might be valid. But when behavioral differences are at issue, all other factors are seldom equal.

5.2 Relativity and the quest for unequivocalness

As was pointed out in section 2.2, Öhman and Dimberg (1984) proposed that phobias are the result of a "relatively" closed genetic program. In this view, it is the convergence of a genetically represented learning capacity and a few actual learning experiences that are responsible for the development of phobias. Recalling that Öhman and Dimberg gave a detailed description of the way in which closed genetic programs function, this is an acceptable hypothesis about the relationship between learning and genetics in the etiology of phobias. However, a serious objection that can be raised is that Öhman and co-workers do not adhere to their own description. In 1986, animal fears suddenly appear to be controlled by open genetic programs (Öhman, 1986; p. 128). What are we supposed to make out of this? Obviously, if the constructs of open and closed genetic programs are employed so loosely, it becomes difficult to see what their usefulness is anyway.

Indeed, the theoretical papers by Öhman et al. now and then suffer from a lack of discipline to use concepts in a consistent way. Particularly germane to this issue are the following quotations:

"And what may be transferred between generations in this way is not learning, but a capacity for it" (Öhman & Dimberg, 1984; p. 57).

"Efficient escape and avoidance responses activated by reptilian stimuli, therefore, must have been intensely selected for in the early evolution of mammals. Consequently, it must have been coded deeply inside the primitive brains that ultimately were to evolve into the enormously complicated and sophisticated machinery that present-day humans carry around inside their skull" (Öhman et al., 1985; p. 19).

"According to the biologically inspired analyses (..), the former set of situations (snakes, spiders etc., H.M.) provided threats to the well-being of pretechnological man or his mammalian predecessors (...), and so have become represented in the human gene pool as an enhanced capacity to become associated with fear and avoidance" (Öhman, 1987; p. 31).

It should be noted that these three quotations contain three different hypotheses about the role of genetics in the etiology of fear. First, it is a general learning capacity, then it becomes an avoidance response tied to reptilian stimuli and still later it is the specific capacity to associate fear with phylogenetic danger stimuli which is genetically coded. Of course, in themselves, these hypotheses are neither logically false nor empirically incorrect. Yet, they bear witness to the inconsistent way in which Öhman and co-workers present their version of the preparedness concept.

5.3 Triangles and interspecific behavior

The least that can be expected of scientists is that they take their very own definitions seriously. Öhman et al. (1985) distinguish between non-communicative, interspecific, and intraspecific behavior (section 2.3). They suggest that fear should be studied as a function of these three categories. They then go on to complain about the earlier ex-

perimental work on fear that ignored these different categories.

> "Psychologists' analyses of fear, for example, have relied heavily on studies of the acquisition of avoidance responses in shuttle boxes, that is to say, they have sampled from the domain of noncommunicative behavior. To what extent can knowledge generated within this particular paradigm tell us anything about fear in the context of communicative behavior, as in predatory and social fears?" (Öhman et al., 1985, p. 14/15).

> "If fear and anxiety originate in different behavioral systems, is it then reasonable to use the former as a model for the latter, as is often done by behavior therapists?" (Öhman, 1987, p. 28).

So far so good. But let us now once again consider the type of study carried out by Öhman et al. (section 3.4, Table 2). They compared SCRs conditioned to slides of snakes and spiders to SCRs conditioned to slides of houses (e.g., Öhman, Eriksson & Olofsson, 1975), circles, triangles (e.g., Hugdahl & Öhman, 1977), mushrooms, flowers (e.g., Hugdahl & Öhman, 1980) or electric outlets (e.g., Hugdahl & Kärker, 1981). In other words, they compared a response directed at elements from the interspecific category to one directed at elements from the noncommunicative category, which is, according to their own analysis, a major mistake. Following this analysis, more appropriate experiments would have been based on a comparison between responses conditioned to snakes or spiders and those conditioned to, say, rabbits or butterflies.

A second, more fundamental point to be noted is the sheer arbitrariness with which Öhman et al. (1985) relate animal fears to the predatory defense system and social fears to the social submissiveness system (section 2.3). Assuming that people can choose for either fight or flight when confronted with a predator or a dangerous conspecific creature, why should evolution have capitalized on flight? Öhman et al. fail to specify why the cost/benefit ratio has shaped the predatory defense and social submissiveness systems in such a way that they produce fear rather than aggression. As it stands, their line of argument can equally well be

used to go for the hypothesis that counterphobic behavior of, for example, tarantula enthusiasts (Kleinknecht, 1982) stems from the predatory defense system while the sociopathic behavior of some criminals is a product of the social submissiveness system. The problem we are facing here is that preparedness as a scenario for the biological advantage of fear behavior does not meet one of the most important requirements for an evolutionary theory, namely that it should be based on an explicit comparison between the outcomes of different scenarios. As Kitcher (1985) expressed it, "Darwinian histories win their way to the top through the elimination of rivals" (p. 66).

Öhman and Dimberg (1984) write:

> "while the difficulties in submitting many of the propositions in this chapter to direct empirical tests would be embarrassing if our aim was theoretical rather than metatheoretical, it is to miss the point to apply this type of criticism, because as metatheory, it is evaluated primarily in terms of its fruitfulness in prompting new theoretical developments and research" (p. 76).

One wonders whether Öhman and Dimberg's choice to theorize at a meta-level is somehow related to the inconsistencies that cropped up in their subsequent theoretical papers (Öhman et al., 1985; Öhman, 1986). Because of its looseness and open-endedness, a metatheoretical formulation allows for theoretical inconsistencies and empirical results that seem difficult to account for. Thus, a metatheoretical formulation can promote a state of theoretical and empirical stagnation. Psychoanalysis provides an extremely good example of this. In fact, it is highly questionable whether psychology is in need of metatheories as long as real theories are scarce.

CHAPTER SIX

PREPAREDNESS AND ITS EMPIRICAL BASIS: A CRITICAL DISCUSSION

In a thorough historical analysis, Harris (1979) points out that Watson and Rayner's "little Albert" experiment (1920) "is rediscovered every 5 or 10 years by a new group of psychologists" (p. 155). He refers to Seligman's article (1971) on preparedness and phobias as a case in point. In this article, "little Albert" is used as an empirical illustration of prepared conditioning while the infant conditioning study by English (1929) is qualified as an instance of unprepared conditioning. As will be remembered, the former study succeeded in establishing a conditioned fear response whereas the latter study failed to do so.

In both studies, loud noise served as the UCS. But whereas Watson and Rayner (1920) used a rat as a CS, English (1929) used a wooden duck as a CS. Seligman (1971) suggests that the superior conditioning found in the former study is due to the prepared character of the rat ("furry things")-noise contingency. Admittedly, Harris' (1979) critical examination of the way in which Seligman represents the "little Albert" study is not entirely convincing (see Seligman, 1980). Yet, in one particular respect it is perfectly right; it makes no sense to compare the "little Albert" experiment to English' study, simply because the noise UCS elicited fear in little Albert but was totally ineffective (i.e., did not evoke any observable UCR) in the English' study (see also Delprato, 1980).

In a reply, Seligman (1980) de-emphasizes the role of the "little Albert" study in his argumentation by arguing that it was used "only slightly and in passing" (p. 215). He adds that, since that time, "a good bit of laboratory evidence" (p. 215) has been accumulated to support the preparedness hypothesis. But has it? The main purpose of the present chapter is to show that this evidence leaves much to be desired. To this end, the studies presented in chapter 4 are placed in a broader context by relating them to the numerous failed replications and disappointing cross-validations in other laboratories.

6.1 Is the fear of God prepared?

Some authors have referred to the non-random distribution of fears as being the "core" (De Silva et al., 1977; p. 76) or the "central datum" (Öhman, 1979; p. 109) of the preparedness hypothesis. Community studies (Agras et al. 1969; Costello, 1982) as well as fear surveys among students (Bennett-Levy & Marteau, 1984) have clearly shown that some stimuli are more frightening than others. There is also evidence to back up the assumption that, in some cases, the selectivity of fears is related to stimulus characteristics such as strange appearance, unpredictability etc. (section 4.1). Thus, altogether, the selective distribution of fears is a well-established fact. However, an interpretation of this fact in terms of evolutionary preparedness is hindered by the following considerations. First, it must be recognized that the number and type of stimulus items that are included in a fear survey are likely to affect the results. For example, Agras et al. (1969) employed only 40 items. These items were derived from traditional fear scales (e.g., Wolpe & Lang, 1964). The fear survey study by Kirkpatrick (1984) was based on 133 items. In addition, the latter study inquired about fears other than those among the items, a procedure that Agras et al. probably did not follow. Thus, it is not surprising to find that snake fear was the most frequent fear in both men and women in the survey by Agras et al., whereas in Kirkpatrick's survey, snake fear ranked 6th and 21st for women and men respectively. Kirkpatrick reported that fear of roller-coasters and fear of untimely death were the top intense fears for women. For men, the fear of being punished by God ranked highest. It is obvious that when the job is to account for the fear rank order found by Agras et al., the preparedness interpretation might be a good guess. But, unless one manages to show that, say, the fear of God can be traced back to some "phylogenetically-relevant" avoidance, a preparedness interpretation of the rank order found by Kirkpatrick is far from satisfactory.

A second point that can be raised is concerned with the validity of the preparedness interpretation. A preparedness interpretation is premature, even if one starts from an ideal fear rank order in which, for example, fear of snakes, enclosed places etc. is far more prevalent than fear of mushrooms, flowers etc.. As long as it has not been established

independently that the former class of stimuli is more "phylogenetically survival relevant" than the latter category, arguments about the preparedness of highly frequent fears remain circular. As was pointed out earlier (section 4.2), there is evidence which suggests that a preparedness interpretation of the ideal rank order is neither the only nor the most powerful explanation. If the "ontogenetic" dangerousness and unpredictability of the feared stimuli are taken into account, a substantial part of the non-random fear distribution can be explained.

A minor point of interest is formed by Öhman's suggestion that the universally found negative connotations of snakes and reptiles are due to a phylogenetically shaped archetype. A number of cultural phenomena cast doubts on this suggestion. To be sure, a lengthy treatise would be required to list all the virtues that have been associated with the snake symbol among many cultures. To name but a few examples, the staff of Aesculapius represents medical skills, images of serpents can be found in the arms of many an aristocratic family, and the snake is a salient detail of the Alfa Romeo vignette. Similarly, inconographic texts (e.g., Kroll & Witte, 1921; Whittick, 1960; Cirlot, 1962) invariably indicate the double denotation of the snake symbol, with negative connotations going hand in hand with a series of virtues.

6.2 The ambiguity of a choking phobia

McNally (1986) described the interesting case of man with a choking phobia. This man had acquired his phobia when he panicked while choking on a piece of fish. For those who are familiar with the preparedness hypothesis it will not be too difficult to identify an adaptational feature of the choking phobia. One could, for instance, point out the fact that the patient avoids potentially dangerous foods. Those who are unfamiliar with the preparedness hypothesis will probably ignore this feature and focus on the fact that a choking phobia is associated with severe weight loss and deteriorated health. This implies that, apart from phobias that seem highly prepared and phobias that seem highly unprepared (section 3.2), there is a third category. This category consists of phobias that are either prepared or unprepared, depending on the characteristics that are singled out and judged.

Clinical studies that are concerned with the prevalence of prepared fears have yielded mixed results (sections 3.2, 4.3). Three out of four studies found a preponderance of highly prepared fears in samples of phobic and obsessional out-patients (De Silva et al., 1977; Zafiropoulou & McPherson, 1986; De Silva, 1988). One study failed to replicate this result in a sample that mainly consisted of obsessive-compulsive in-patients (section 4.3). It could be argued that this failure to replicate earlier results is due to patient characteristics such as the severity of the disorder, the presence of obsessive-compulsive symptoms etc.. It should be noted, however, that the preparedness hypothesis assumes that most fears, including those of obsessive-compulsive patients, are prepared (De Silva et al., 1977). Furthermore, it assumes that there is a positive correlation between severity, as defined by resistance to extinction, and preparedness (De Silva et al., 1977). Although the incidental occurrence of unprepared phobias is not ruled out (Rachman & Seligman, 1974), one would expect on the basis of these assumptions to find a vast majority of severe fears to be prepared.

As the example of the choking phobia illustrates, clinical fears can be broken down into a series of characteristics (e.g., feared stimulus, avoidance, consequences etc.). Subsequent selection of one or several of these characteristics is likely to affect the amount of preparedness that a judge will attribute to the clinical fear under consideration. The divergent results of clinical studies might be related to this selection process. Direct evidence to support this view is lacking. But it is striking that De Silva et al. (1977) asked judges who were highly familiar with the preparedness concept to rate relatively extensive clinical descriptions, whereas the study presented in section 4.3 asked judges who were unfamiliar with this concept to rate feared stimuli rather than descriptions. Thus, it may well be that the high frequency of prepared phobias reported by De Silva et al. (1977) and by studies that were conducted along similar lines (Zafiropoulou & McPherson, 1986; De Silva, 1988) is a by-product of the rating procedure. All in all, it remains to be seen whether "the great majority of phobias are about objects of natural importance to the survival of the species" (Seligman & Hager, 1972; p. 460).

A second issue to be noted is that the clinical studies that were carried out so far, agree on one important point: None of them was able to show that prepared fears are characterized by a sudden or early age of onset. On the contrary, there is some evidence that the more a fear is prepared, the later its age of onset is (section 4.3). Given the fact that sudden or early onset is considered to be the defining characteristic of prepared learning, these findings are a severe blow to the empirical status of the preparedness hypothesis.

A third point is the clinical evidence for the notion that both animal and social phobias are the outcome of prepared conditioning. As for animal phobias, there are, indeed, a number of psychophysiological studies that suggest that in the case of animal phobics, slides of, for example, snakes or spiders act as CSs for all kinds of physiological responses (Prigatano & Johnson, 1974; Hare & Blevings, 1975a; 1975b). However, the evidence for the idea that social phobias can be pinpointed to a fear of angry facial expressions is far from encouraging (section 4.4). Some investigators might argue that, according to Öhman's theoretical framework, social phobias are less clearly associated with autonomic activation than animal phobias. However, the mere fact that social phobics and normal control subjects do not differ with regard to the subjective evaluation of angry faces casts doubts on the assumption that these stimuli represent a CS for social fears.

A final point that remains to be discussed is Öhman et al's (1985) proposal that both sympathetic activation and automatic (i.e., preconscious) stimulus processing are more strongly present in animal than in social phobias. As for the role of sympathetic processes, they reported that a heart rate acceleration can be demonstrated more easily in animal phobics who are confronted with their feared stimulus (i.e., snakes, spiders, rats) than in social phobics who are confronted with a critical task (i.e., conversation with a member of the opposite sex).

One possible objection to accepting this finding as a confirmation of the above-mentioned proposal is that it might be a product of the testing procedure. That is to say, the tasks used by Öhman et al. (1985) for eliciting fear in animal and social phobics are not matched. It is obvious that animal phobias are much more focussed on a single situation than social phobias are. As a result, the **mise en scene** of an effective

exposure for social phobias will be much more difficult. It is highly unlikely that fear of a conversation with a member of the opposite sex is the primary fear in most social phobics.

The second problem with respect to the predictions formulated by Öhman et al. is that they seem to be at variance with Lader's research (1967; 1980). Lader found that spontaneous electrodermal fluctuations and SCR magnitudes are higher in social phobics than in animal phobics, whereas habituation rates tend to be faster in animal phobias than in social phobias.

As for the alleged difference between animal and social phobics in automatic stimulus processing, direct experimental evidence is lacking. Yet, it is striking that in his recent research, Öhman (1986; Öhman, Esteves, Parra & Soares, 1988) reported that angry face CS+s elicit conditioned SCRs when they are presented for only 30 milliseconds and are then followed by a mask, whereas happy face CS+s do not. Short stimulus exposure combined with backward masking prevents conscious recognition of stimuli and, consequently, provides a reliable method for studying pre-attentive processes (Marcel, 1983). In so far as a conditioned SCR to angry faces can be taken as a forerunner of social fears (Öhman & Dimberg, 1984), the recent data collected by Öhman suggest that pre-attentive processing, at least, plays a part in these fears.

6.3 Creating an illusory correlation with the aid of the Beatles

In his classical conditioning studies, Rescorla (1968) showed that the strength of the CR is directly related to the predictive value of the CS in signalling the occurrence of a UCS. According to Rescorla's model of classical conditioning, the predictive value of the CS is a function of objective facts such as the number of UCS-alone trials and combined CS-UCS trials. With their illusory correlation paradigm, Mineka and Tomarken (1989) have taken this model one step further. Their data strongly suggest that the predictive value of the CS is also greatly affected by the content of this CS. Despite the fact that the percentage of CS-shock, CS-alone, and UCS-alone trials was exactly the same for neutral and phobia-relevant stimuli in their experiments, subjects attached more predictive value to the phobia-relevant stimuli than to the

neutral ones (section 3.3). Mineka and Tomarken (1989) have undoubtedly found an interesting phenomenon. Nevertheless, their attempt to relate this phenomenon to the etiology and maintenance of prepared fears is problematic. Their failure to examine the effect of "ontogenetically dangerous" CSs on the illusory correlation phenomenon is a major obstacle to this attempt. Only an experiment in which phobia-relevant CSs (e.g., snakes, spiders), neutral CSs (e.g., mushrooms, flowers), as well as "ontogenetically dangerous" CSs (e.g., rifles pointed at the subject; section 6.6) are employed can answer the question whether the illusory correlation phenomenon is linked more strongly to phobia-relevant stimuli than to all other types of stimuli.

Although the parallel with the taste aversion studies urges itself on us, the illusory correlation phenomenon **per se** does not imply that survival-relevant associations are involved. This is nicely illustrated by Karmel's experiment (cited in Seligman & Yellen, 1987). In this experiment some 50 christmas light bulbs blinked on and off in a chaotic, random order. But as soon as music by the Beatles was presented simultaneously, subjects reported that, in their perception, the christmas lights seemed to follow the rhythm of the music. This is a straightforward example of a bias that results from the salience of the stimuli rather than from their survival relevance. Similarly, it would not be surprising at all to find that the biases reported by Mineka and Tomarken are related to the attention-attracking properties of phobia-relevant stimuli.

6.4 Habituation in conditioning studies: Preconditioning effects complicate interpretations

Though untestable, it is at least plausible to assume that Seligman's hypothesis about preparedness and phobias (1971) would have sunk into oblivion but for Öhman's research. This is indicated by the fact that in recent texts dealing with the experimental study of anxiety, Seligman's hypothesis and Öhman's research are virtually always bracketed together (e.g., Eysenck, 1979; Marks, 1987; Mineka, 1985). The general purport of many of these texts is that although the preparedness hypothesis is a bold proposal, it has obtained a **raison d'être** by means

of Öhman's research. Marks (1987), for example, writes

> "That we perceive, learn, and do what comes naturally has not been easy to demonstrate conclusively in the laboratory. A pioneer step in this regard is the "differential conditioning" paradigm of Öhman and his co-workers" (p. 232).

Yet, what is generally overlooked or, at least, underestimated are the serious difficulties both in interpreting and replicating the phenomena reported by Öhman. The most appropriate way to list these difficulties is to relate them, one by one, to the habituation, acquisition, and extinction phases of the conditioning procedure.

In turning now first to the habituation phase, Gray's (1982) re-interpretation of Öhman's results (section 3.4) deserves mentioning. Basically, this re-interpretation holds that preparedness is a superfluous concept if the simpler move "to treat spiders and snakes and the like as innate stimuli for fear, requiring no conditioning at all" is chosen (Gray, 1982; p. 434). Gray believes that SCRs to phobia-relevant CSs reflect innate fears which are further potentiated by electric shocks and, hence, become resistant to extinction. This would imply that a preconditioning difference in emotional impact of the CS rather than an interaction of conditioning and heredity is responsible for fear-relevance effects during extinction. Some authors (e.g., Rachman, 1978; Bennett-Levy & Marteau, 1984) have treated the preparedness hypothesis and Gray's interpretation as quite similar formulations. There is, however, a crucial difference. In Pavlovian terms, this difference could be summarized as follows: Whereas the preparedness hypothesis regards stimuli such as snakes and spiders as members of a special subclass of CSs, Gray assumes that such stimuli function as UCSs for anxiety.

On the basis of Gray's view, orienting responses to phobia-relevant stimuli would be expected to be stronger and to habituate more slowly than those to neutral stimuli. On the other hand, the fact that a stimulus elicits strong and slowly habituating orienting responses does not necessarily imply that it mobilizes innate fears. Although a prepotency effect during habituation was only occasionally found in Öhman's studies (e.g., Öhman, Eriksson & Olofsson, 1975; section 3.4,

Table 2), there is something to be said for Gray's interpretation.

A potential problem of Öhman's conditioning studies as to evaluating preconditioning differences is that they involve only a small number of habituation trials (usually 4 for each type of CS) and are often based on a single dependent variable, namely the SCR. It is interesting to note that in one of their few electrodermal habituation studies, Öhman and co-workers (Öhman, Eriksson, Hugdahl & Olofsson, 1974; Exp. 1, Exp. 2) found that phobia-relevant stimuli (i.e., snake pictures) elicit stronger (Exp. 2) or more slowly habituating (Exp. 1, Exp. 2) responses than neutral stimuli (i.e., pictures of houses). This study employed 10 or more habituation trials. A study by Dimberg (1986b) is also relevant to the issue under consideration. This author reported that phobia-relevant (i.e., snakes or spiders) and neutral (i.e., mushrooms, flowers) stimuli evoke completely different EMG response patterns: Phobia-relevant stimuli were associated with increased corrugator activity, whereas neutral stimuli were associated with increased zygomatic activity. Similarly, the data presented in section 4.4 indicate that both in normals and in social phobics, angry face stimuli give rise to larger SCRs and stronger EBR inhibition than happy face stimuli.

What can be concluded from these studies is that under certain conditions (e.g., extensive habituation procedure, the use of other dependent variables than the SCR), preconditioning differences between phobia-relevant and neutral stimuli do emerge. The question that remains is: To what extent do these differences support Gray's interpretation? In other words, do these differences indeed indicate that phobia-relevant stimuli function as UCSs whereas neutral stimuli function as CSs? In order to investigate this issue, Merckelbach, van den Hout, and van Oppen (1988) carried out a conditioning procedure (N=23) in which one neutral slide (CS+) was paired with a phobia-relevant stimulus (US)[1], while another neutral slide (CS-) was paired with a third, neutral control stimulus (NCS). The CS-\NCS pairings were used to control for pseudo-conditioning effects. Pictures of mushrooms, flowers, or apples served as

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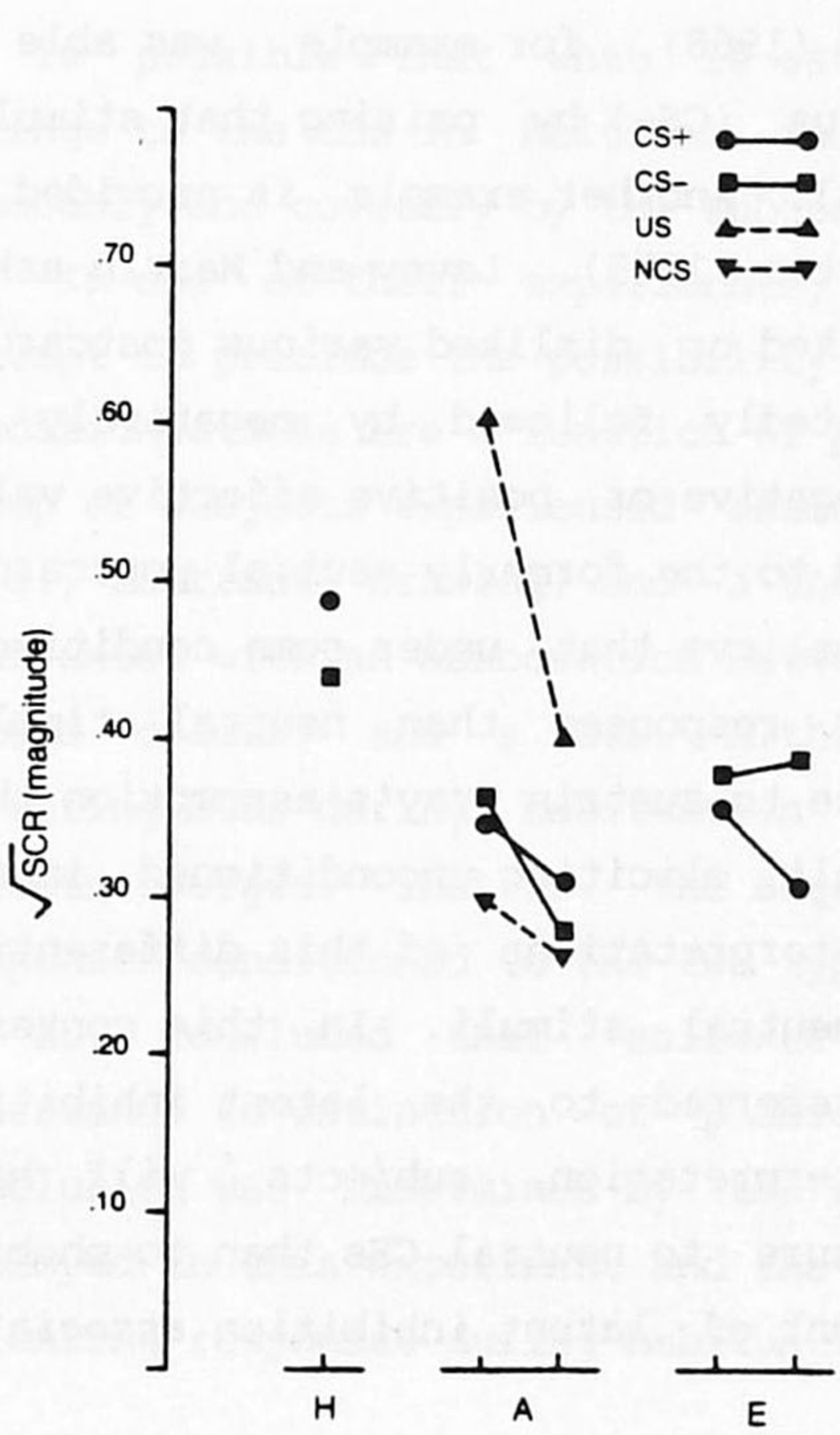
[1] The abbreviation US is used here instead of UCS to emphasize that the phobia-relevant slides are employed as unconditioned stimuli in a technical sense. It is an empirical matter whether these stimuli are aversive enough to elicit successful conditioning.

CS+, CS-, or NCS. Slides of snakes or spiders were used as US. The experiment was based on two dependent variables. First, SCRs to the stimuli were recorded. Second, subjects rated CS+, CS-, NCS, and US in terms of pleasantness on a 14 cm visual analog scale, ranging from 0 ("extremely unpleasant") to 14 ("extremely pleasant"). These ratings were obtained both before ("pre") and after ("post") the conditioning procedure. It was found that the phobia-relevant slides (USs) elicited greater orienting SCRs and were evaluated more negatively than the neutral slides (CS+, CS-, NCS). However, no evidence was found to suggest that the phobia-relevant slides actually functioned as unconditioned stimuli. Thus, the CS+ did not evoke increased SCRs as a result of its pairing with a phobia-relevant stimulus (Figure 1).

Figure 1. MEAN √SCR TO CS+ AND CS- AS A FUNCTION OF HABITUATION (H), ACQUISITION (A), AND EXTINCTION (E) TRIALS. MEAN √SCR TO US AND NCS DURING ACQUISITION ARE ALSO SHOWN (FROM MERCKELBACH, VAN DEN HOUT & VAN OPPEN, 1988).



Neither was there a significant difference between the pre- en post-conditioning evaluation of the CS+. That is, the CS+\US pairing did not result in a significantly more negative evaluation of the CS+ (Table 1).

Table 1. MEAN SUBJECTIVE EVALUATIONS (0-14) OF PREPARED UNCONDITIONED STIMULI (US), NEUTRAL CONDITIONED STIMULI (CS+, CS-), AND NEUTRAL CONTROL STIMULI (NCS) BEFORE (PRE) AND AFTER (POST) THE CONDITIONING PROCEDURE. STANDARD DEVIATIONS ARE GIVEN BETWEEN PARENTHESES (FROM MERCKELBACH, VAN DEN HOUT & VAN OPPEN, 1988).

	US	CS+	CS-	NCS
PRE	4.2 (2.9)	9.9 (2.5)	10.1 (2.7)	11.2 (2.3)
POST	4.0 (2.1)	9.6 (2.4)	9.7 (2.4)	11.4 (2.1)

In order to place this experimental attempt at verifying Gray's assumption in an appropriate perspective, it should be remembered that the conditioning literature offers many illustrations of successful conditioning with non-painful UCSs. Geer (1968), for example, was able to condition SCRs to an auditory stimulus (CS+) by pairing that stimulus with photographs of violent death (UCS). Another example is provided by evaluative conditioning (Levey & Martin, 1975). Levey and Martin asked subjects to indicate how much they liked or disliked various postcards. When neutral postcards were repeatedly followed by negatively or positively evaluated postcards, the negative or positive affective value of the later postcards was transferred to the formerly neutral postcards.

In sum, there are reasons to believe that, under some conditions, phobia-relevant stimuli evoke stronger responses than neutral stimuli. However, there is, as yet, no evidence to sustain Gray's assumption that this is due to phobia-relevant stimuli eliciting unconditioned, innate fear. There are more plausible interpretations of this differential responding to phobia-relevant and neutral stimuli. In this context, McAllister and McAllister (1979) referred to the latent inhibition phenomenon. According to their interpretation, subjects will have experienced more non-reinforced exposure to neutral CSs than to phobia-relevant CSs. Consequently, the amount of latent inhibition associated

with neutral CSs will be greater and this, in turn, might reduce orienting responses to neutral CSs. Another possibility was advanced by Maltzman and Boyd (1984). In two experiments these authors demonstrated that the differential electrodermal responding to phobia-relevant CSs (i.e., snakes, spiders) and neutral CSs (i.e., mushrooms, flowers) becomes particularly strong after subjects have been asked to rate the significance of these stimuli on a semantic differential scale. At the same time, their rating data showed that phobia-relevant stimuli are evaluated as more unpleasant than neutral stimuli. These investigators suggested that pictures of snakes and spiders are more significant or interest-attracking than pictures of mushrooms and flowers. They rightly added that an interest-attracking property is a much broader quality than phobia relevance, and concluded that

> "Assigning signal value to the stimuli by requiring their overt ratings appears to have a multiplicative effect on pre-existing differences in their significance" (p. 45).

It is possible that what is established by the instruction to assign ratings to the CSs in Maltzman and Boyd's experiment, is done spontaneously and covertly by the subjects in the typical Öhman studies.

In one of their experiments, Öhman et al. (1976, Exp. 2) made an attempt to preclude the possibility that differential extinction rates of conditioned SCRs are a function of pre-existing stimulus differences. One group of subjects experienced associations between a complex stimulus (i.e., abstract drawing) and a shock UCS. A second group was repeatedly confronted with an association between a simple stimulus (i.e., background colour) and a UCS. Although the complex CS elicited stronger orienting SCRs during habituation than the simple CS, no carry-over effects emerged. That is, the acquisition and extinction curves of the responses conditioned to the two types of stimuli did not differ. Öhman et al. concluded that "salience is not a factor behind the superior resistance to extinction of phobic stimuli" (p. 326). However, this conclusion was undermined by the relative insignificance of the stimuli employed in this experiment and the quick disappearance of differential orienting responses during habituation.

Latent inhibition of neutral CSs or interest attracking properties of phobia-relevant CSs, whichever of the two turns out to be the correct interpretation of the differential responding to these stimuli, it is clear that preconditioning differences make a straightforward interpretation of subsequent acquisition or extinction effects during a conditioning procedure rather difficult. As it is likely that CS salience affects conditioning[2], it can not be ruled out that the effects reported by Öhman, at least partly, have a non-associative basis.

6.5 Acquisition in conditioning studies: The disregard of a refuted prediction

The quotation (section 3.1) from Seligman's 1970 article that was presented earlier does not leave any doubt about the defining feature of prepared learning. The definition of prepared learning relies explicitly on the criterion of "ease of acquisition". Delprato (1980) and McNally (1987) rightly remarked that the results reported by Öhman and co-workers rarely meet this criterion. In most of their experiments, they found fear-relevance effects to be limited to the extinction phase. Only occasionally did fear-relevance effects occur during the acquisition phase (section 3.4, Table 2). Adherents of the preparedness hypothesis (e.g., Rachman, 1978; Öhman, Fredrikson & Hugdahl, 1978b) have made a perilous move in order to deal with this problem. Instead of concluding that most of the time Öhman's acquisition data are not indicative of prepared learning, they proposed to redefine the concept of preparedness in such a way that it will accord with Öhman's results. Thus, Rachman (1978) writes

> "For intrinsic reasons, and because it offers the promise of much wider applicability of the theory, these findings support the idea that the main defining feature of biologically relevant phobic stimuli might preferably rest on persistence rather than rapid acquisition" (p. 236).

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[2] For example, on the basis of animal data, Rescorla and Wagner (1972) proposed a widely accepted formalization of Pavlovian learning in which the associative strength of a CS does not only depend on UCS characteristics but also on the salience of the CS.

In the light of the numerous studies that failed to find a persistence of SCRs conditioned to phobia-relevant stimuli during extinction (section 6.6), Rachman's redefinition is premature, to say the least. As long as the empirical status of Öhman's findings is unclear, it does not make any sense to accept these findings as paradigmatic examples of prepared learning on the basis of which the definition of preparedness might be accentuated. Additionally, the lack of fear-relevance effects during the acquisition phase in Öhman's experiments is ignored rather than illuminated by such a redefinition. Indeed, an interpretation of the absence of acquisition effects should not start with rash redefinitions but with technical considerations.

The older conditioning literature leads one to believe that the strength of a conditioned SCR is gradually built up as the result of successive CS+\UCS pairings during acquisition. This is an untenable view. Maltzman (1979) presented strong evidence to support the claim that the subject's discovery of the CS+ as a predictor of the UCS is a necessary prerequisite for successful acquisition of a conditioned SCR. He also showed that this discovery gives rise to a sudden and strong conditioned SCR that reaches an asymptote very early. Furthermore, the persistence of this asymptotic SCR appears to be a function of UCS intensity. In the differential conditioning procedure typically followed by Öhman, it is very easy for the subject to discover which stimulus is followed by a shock UCS. In addition, Öhman usually employed UCSs of a relatively mild intensity ("annoying but not painful"). In the light of the data presented by Maltzman, it is not surprising to find that both conditioned responses to phobia-relevant CS+s and those to neutral CS+s are suddenly acquired, reach a ceiling very soon, and finally diminish. Thus, potential differences between the SCRs to phobia-relevant CS+s and those to neutral CS+s are probably overshadowed by ceiling effects. Of course, it is possible to adopt a slightly different attitude towards this issue by concluding that it is perfectly possible to account for Öhman's acquisition data without referring to the concept of preparedness.

Despite the repeated failure to find fear-relevance effects during acquisition, Öhman (1979) tried to bring his findings in line with the "ease of acquisition criterion" by referring to one of his first experiments. In this experiment it was found that one pairing of a

phobia-relevant CS with a UCS during acquisition is sufficient to elicit a fear-relevance effect during extinction. Öhman went on to remark that "it always takes a number of trials to establish conditioned fear in the laboratory" (p. 120) when neutral CSs are employed. While the suggestion that electrodermal conditioning can be equated with fear conditioning is ignored for a moment, it must be noted that Öhman's remark is not justified by a careful examination of the conditioning literature. In this literature some pertinent examples of one-trial conditioning of electrodermal responses to neutral stimuli can be found (Campbell et al., 1964; Pendery & Maltzman, 1977).

6.6 Extinction in conditioning studies: Failures multiply at a fast rate

The delayed extinction of psychophysiological responses conditioned to phobia-relevant stimuli (section 3.4; Tables 2, 3) has been the most systematic finding in the experiments by Öhman and associates. As their experimental results indicated that the delayed extinction is presumably limited to a specific class of stimuli (i.e., snakes, spiders, angry faces) and is not easily instructed away (e.g., Hugdahl & Öhman, 1977), Öhman and co-workers claimed to have developed a laboratory model for the selectivity and non-cognitivity of real life phobias (e.g., Öhman, 1979). This claim is open to serious criticism, both at a theoretical and at an empirical level.

It may be useful to begin our discussion with a number of interpretatory problems that are associated with the fear-relevance effect during extinction. In the first place, the problematic relationship between autonomic variables and fear in Öhman's experimental work should be noted. Some authors take it for granted that the conditioned SCRs in Öhman's experiment reflect fear. Rachman (1978), for instance, looks upon Öhman's experiments as successful demonstrations of the resistance to modification of "prepared fears" (p. 131). Öhman and co-workers themselves are of the opinion that the SCR taps the physiological domain of

fear[3]. Indeed, the very term "fear-relevant" (e.g., Fredrikson & Öhman, 1979) implies that the **modus operandi** behind Öhman's results is fear. Yet, a close examination of the type of SCRs that proved to be sensitive in Öhman's research reveals that this suggestion is misleading. To make this clear, some remarks concerning the SCR are in order.

When, in a conditioning procedure, the CS is presented some eight seconds before the aversive UCS occurs, multiphasic SCRs can be recorded. That is, it is usually possible to distinguish between an early SCR component, which is referred to as the first-interval anticipatory response (FAR), and a late SCR component, which is known as the second-interval anticipatory response (SAR). These components differ as to their temporal and psychological properties (Prokasy & Kumpfer, 1973). The FAR component emerges 1 to 4 seconds after stimulus onset and is said to be an index of orienting or attention towards the CS. The SAR component occurs 4 to 8 seconds after stimulus onset and is usually considered to reflect the anticipation of an impending UCS (see, however, Maltzman, 1987, for an alternative interpretation). Assuming that the classical conditioning procedure parallels the etiology of clinical fears, the SAR component is expected to be the most appropriate experimental analogue to anticipatory fears. In contrast with this, Öhman found fear-relevance effects to be characteristic of the FAR rather than of the SAR component (see review by McNally, 1987). He proposed that his classical conditioning procedure changed the emotional value of the CS and, consequently, the orienting reactions towards the CS (e.g., Öhman et al., 1976; Öhman, 1979). Now, the preparedness hypothesis is, of course, about conditioned fears and not about conditioned attention. But, even if this point is ignored for a moment, it should be noticed that Öhman's proposal leaves the question unanswered why the SAR component turned out to be relatively ineffective for obtaining fear-relevance effects (Gray, 1982).

As for the FPV, it is striking that in Öhman's experiments the interpretation of this measure changes along with its usefulness in

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[3]Some researchers have vigorously argued against such an interpretation. Furedy and Arabian (1979), for example, stated: "When you pair a pain-inducing stimulus like shock with a neutral tone and that pairing produces an increase of GSR, you may think that the GSR reflects conditioned fear, but all it really reflects is increased attention" (p.355).

demonstrating fear-relevance effects. Öhman, Eriksson and Olofsson (1975) recorded both SCR and FPV, but found fear-relevance effects to be limited to the SCR. To account for this discrepancy between the autonomic variables, they proposed that "peripheral vasomotor changes are less sensitive to psychological impact than are electrodermal responses" (p. 625). This proposition is not only at variance with a number of habituation studies that indicated a close relationship between FPV and anxiety (O'Gorman, 1977), but also with the point of view that the Öhman group presented several years later. Fredrikson and Öhman (1979) reported fear-relevance effects during extinction for SCRs as well as for FPVs. Accordingly, they suggested that the fear-relevance effect is a broad, sympathetic phenomenon that also encompasses cardiovascular aspects, in casu, vasoconstriction in the FPV.

Fredrikson and Öhman (1979) and Öhman et al. (1978c) tried to endorse an interpretation of their results in terms of fear by referring to Sokolov's (1963) distinction between orienting and defensive responses. They argued that in an aversive conditioning procedure, phobia-relevant CSs elicit a more or less permanent defensive response whereas neutral CSs elicit a transitory orienting response. However, the empirical evidence that had to cover this theoretical move proved to be equivocal. Fredrikson and Öhman (1979) failed to obtain cardiac acceleration to phobia-relevant CSs and cardiac deceleration to neutral CSs. On the other hand, Öhman et al. (1978c) succeeded in demonstrating palmar vs. dorsal differentiation in SCRs conditioned to phobia-relevant CSs. They suggested that this differentiation could be regarded as an index of defensive responses to this class of CSs. Their suggestion is essentially a speculative elaboration of Edelberg's (1973) theory of effector mechanisms in electrodermal activity. Meanwhile, it should not be overlooked that the discussion about the precise psychophysiological features of the defensive response is still continuing (e.g., Barry & Maltzmann, 1985).

Apart from the question whether it is a physiological aspect of fear that is conditioned in Öhman's procedure, there is a more fundamental objection that can be raised. Öhman and co-workers maintain that the delayed extinction of psychophysiological responses conditioned to phobia-relevant stimuli mimics the persistence of real life phobias. There are, however, good reasons to ascribe the persistence of real life

phobias to successful avoidance of feared stimuli rather than to a failure of phobic responses to extinguish. Thus, it would seem that simple phobias that pertain to survival-relevant stimuli (e.g., snakes, spiders etc.) can be eliminated simply by a therapeutic exposure to these stimuli (McNally, 1987). Having reviewed various treatment approaches and their outcomes, Sturgis and Scott (1984) concluded that there is quite some evidence which suggests that treatment techniques such as implosion and flooding "are highly effective in the treatment of simple phobias" (p. 126). Similarly, Emmelkamp (1983) proposed that effects on social phobics of training social skills

> "may be due to exposure in vivo to social situations. During treatment sessions patients are exposed to anxiety arousing situations and have to give up their avoidance behavior, which eventually may lead to anxiety reduction" (p. 137).

In keeping with this, several clinical studies (section 6.2) failed to document differences in treatment outcome between "prepared" phobias and "unprepared" phobias. Admittedly, one study (section 4.3) found "prepared" obsessions to be associated with a relatively poor outcome, but this finding requires replication. All in all, longitudinal, controlled studies are needed to ascertain whether the critical process underlying the persistence of phobias is formed by successful avoidance or by resistance to extinction of phobic responses per se. Until this issue has been examined thoroughly, Öhman's claim that his results provide an analogue to the persistence of phobias is highly premature.

A final, theoretical point that is worth noting is the flexibility of the preparedness hypothesis as to accounting for a wide range of experimental results. An illustration of this is the way in which advocates of the preparedness hypothesis (e.g., Marks, 1987) interpreted the findings of Lanzetta and Orr (1980).

Using a differential, classical conditioning paradigm, Orr and Lanzetta (1980) found a better acquisition of the conditioned SCR when a fearful facial expression is used as a CS+ than when a happy facial expression is used as a CS+. A subsequent study validated this finding (Lanzetta & Orr, 1980). In this study, a compound stimulus consisting of

a tone combined with either a happy or a fearful face was reinforced by a shock. During test sessions in which tones and faces were presented separately, SCRs to fearful face CS+s exceeded those to the tone, whereas SCRs to happy face CS+s tended to be lower than those to the tone. These results were replicated in two further studies (Lanzetta & Orr, 1981; Orr & Lanzetta, 1984). The investigators concluded that fear expressions have excitatory properties when used as CS+ and, consequently, are able to overshadow the conditioned responses to neutral cues such as tones.

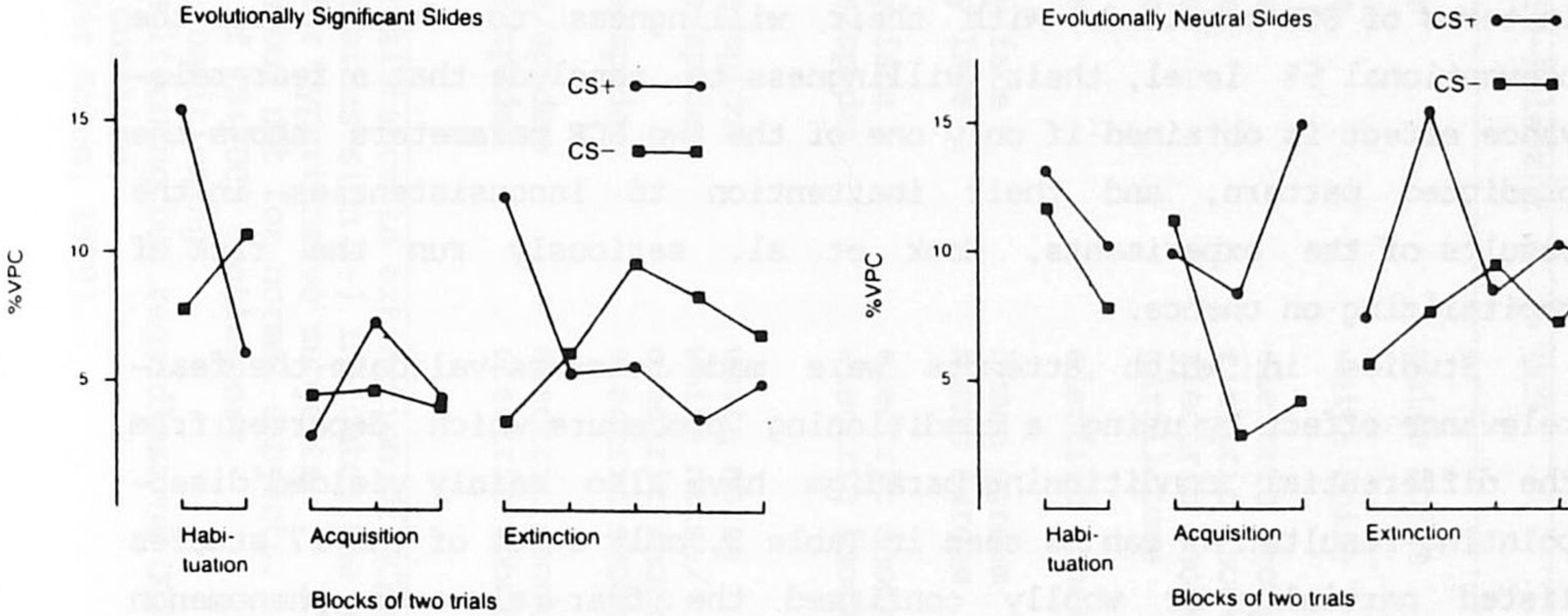
Dimberg and Öhman (1983), Dimberg (1987), and Marks (1987) referred to Orr and Lanzetta's findings as though they are consistent with the results of studies in which angry faces were found to be superior CS+s (section 3.4, Table 3). Recalling that the evolutionary rationale behind Öhman and Dimberg's experiments is the idea that angry faces signal a threatening social conflict, such an interpretation of Orr and Lanzetta's results can be seriously questioned. Following Öhman and Dimberg's reasoning (1984), it is tempting to argue that a fearful face signals a defeat in a social dominance conflict and hence elicits, say, relief or intensified, offensive attack in a conspecific. In other words, it is difficult to imagine how it can reasonably be argued that a fearful face CS+ evokes a fear-relevance effect. It may be objected that a general sympathetic arousal rather than a specific emotion is conditioned in the studies by Öhman and Lanzetta and Orr. Yet, Öhman and co-workers go far beyond such a modest interpretation when they present their conditioning procedure as a model of the etiology of social and animal phobias.

Two points relevant to the empirical weakness of Öhman's model will now be outlined: The lack of empirical robustness of fear-relevance effects during extinction and the alleged non-cognitivity of these effects.

As for the first point, it should be noted that, to date, 6 attempts have been made to replicate the fear-relevance effect using an experimental procedure that comes as close as possible to following the differential conditioning paradigm commonly employed by Öhman and co-workers. Eelen (cited in Emmelkamp, 1982), McNally and Foa (1986), and the experiment presented in section 4.5 have all failed to trace fear-relevance phenomena. In the last mentioned experiment, FPVs were also recorded, in addition to SCRs. The FPV data were discussed in a separate paper (Merckelbach, Jansen, van den Hout & van der Molen, 1987).

Vasoconstriction in the FPV (i.e., volume pulse change; VPC) was quantified by means of the criteria of Furedy (1968). As can be seen in Figure 2, the acquisition procedure did not yield stable vasoconstriction responses to the CS+s. As a result, fear-relevance effects during extinction did not occur. Interestingly enough neutral CS+s were associated with a stronger vasoconstriction than phobia-relevant CS+s during acquisition, a result that strongly deviates from the findings reported by Fredrikson and Öhman (1979).

Figure 2. MEAN PERCENTAGE VOLUME PULSE CHANGE (VPC) IN THE FPV AS A FUNCTION OF TRIAL BLOCKS TO REINFORCED (CS+) AND NONREINFORCED (CS-) STIMULI (FROM MERCKELBACH, JANSEN, VAN DEN HOUT & VAN DER MOLEN, 1987).



The fourth study to be mentioned deserves special attention. Johnsen and Hugdahl (1988) examined the vexed question whether "ontogenetic factors" might be responsible for fear-relevance effects (section 3.4; Hugdahl & Kärker, 1981). The most critical comparison made in their study was that between SCRs conditioned to phobia-relevant CS+s (i.e., snakes) and SCRs conditioned to "ontogenetic dangerous" CS+s (i.e., guns pointed at the subject). The results presented by Johnsen and Hugdahl unequivocally demonstrate that SCRs to the latter category of CS+s extinguish more slowly than those to the former category of CS+s. This implies that "ontogenetically dangerous" CS+s can elicit fear-relevance effects and, hence, it is conceivable that the findings of Öhman et al. can be attributed to the "ontogenetic dangerousness" of phobia-relevant CS+s.

Using an electric shock as the UCS and snakes, spiders, mushrooms, or flowers as CS+s, Cook et al. (1986; Exp. 5, Exp. 6) "enthusiastically" (p. 204) reported a fear-relevance effect during extinction. Nevertheless, a close inspection of their data reveals that there is no reason for this enthusiasm. For example, of the two SCR indices (magnitude and probability of the FAR SCR) that were computed in experiment 5, there was no fear-relevance effect on the probability index. Furthermore, the F-value on the magnitude parameter did not reach the conventional 5% level ($p<0.08$). Experiment 6 yielded completely different results. This time, the non-significant trend of experiment 5 as to SCR magnitude was not replicated. Instead, a significant effect on the SCR probability appeared. These inconsistencies are quite puzzling, particularly when it is borne in mind that Öhman and co-workers reported most of their results in terms of SCR magnitude. With their willingness to depart from the conventional 5% level, their willingness to conclude that a fear-relevance effect is obtained if only one of the two SCR parameters shows the predicted pattern, and their inattention to inconsistencies in the results of the experiments, Cook et al. seriously run the risk of capitalizing on chance.

Studies in which attempts were made to cross-validate the fear-relevance effect by using a conditioning procedure which departed from the differential conditioning paradigm have also mainly yielded disappointing results. As can be seen in Table 2, only 6 out of the 17 studies listed partially or wholly confirmed the fear-relevance phenomenon during extinction. Considering the number of failed replications (4), half-hearted successes (2), and disappointing cross-validations (11), it is hard to escape the conclusion that the fear-relevance effect of phobia-relevant stimuli is, at best, a fragile and small-scale phenomenon that can easily be overruled by as yet unknown factors.

Cook et al. (1986) suggested that one such factor might be the "belongingness" between CS+ and UCS. In their first three experiments, they used a noise UCS and failed to observe fear-relevance effects (Cook et al., 1986; Exp. 1 through 3). In their sixth study, they directly compared the effectiveness of shock and noise UCSs and reported that fear-relevance effects are restricted to subjects who had received a shock UCS. Cook et al. argued that the "belongingness" between a snake or

Table 2. AN OVERVIEW OF ELECTRODERMAL CONDITIONING STUDIES WHICH ATTEMPTED TO CROSS-VALIDATE THE FEAR-RELEVANCE EFFECT.

Study	Phobia-Relevant CS+	UCS	Experimental Paradigm	Fear-Relevance Effect *1
Emerson & Lucas (1981)				
Experiment 1	snake	white noise	one-trial compound-signal conditioning	-
Experiment 2	snake	white noise	three trials compound-signal conditioning	-
McNally & Reiss (1982)	snake	electric shock	Safety-signal conditioning	-
Deitz (1982)	snake	electric shock	single cue conditioning	-
McNally & Reiss (1984)	snake	electric shock	safety-signal conditioning	-
Eifert & Schermelleh (1985)	snake	shrill tone	language conditioning during extinction	+
McNally (1986)	snake	electric shock	one-group differential conditioning	-
Vaitl, Gruppe & Kimmel (1986)	angry face	human scream	transswitching	-
Hamm & Vaitl (1986)	angry face	human scream	one-group differential conditioning	+*2
Pitman & Orr (1986)	angry face	electric shock	instructed extinction	+*3
Dawson, Schell & Tweddle Banis (1986)	snake/spider	electric shock	masking of CS+/UCS contingency	+*4
Siddle, Bond & Friswell (1987)				
Experiment 1	snake/spider	white noise	second order conditioning	-*5
Experiment 2	snake/spider	white noise/ electric shock	second order conditioning	+*6
Björkstrand (1987)	snake/spider	electric shock	CS pre-exposure	-
Foa, Steketee, McCarthy, McNally & Grayson (1988)	snake/spider	electric shock	shock avoidance paradigm	+*7
Merckelbach & van den Hout (1988)	snake/spider	electric shock	trace conditioning	-

*1 A plus sign (+) indicates greater resistance to extinction of SCRs conditioned to phobia-relevant CS+s than of SCRs conditioned to neutral CS+s. A minus sign (-) indicates the absence of such a fear-relevance effect.
*2 For normotensive subjects but not for hypertensive subjects.
*3 For patients with anxiety disorders but not for normal subjects.
*4 Only for subjects who became aware of CS+/UCS contingency.
*5 For neither first nor second order conditioning.
*6 For both first and second order conditioning but regardless of UCS type.
*7 For 5 sec. but not for 10 sec. CS+ duration.
(From Merckelbach, van den Hout & van der Molen, 1989).

spider CS and a shock UCS is quite great since an encounter with a snake or spider may have an aversive, tactile effect, just as a shock UCS does.In contrast with this, the "belongingness" between a snake or spider CS and a noise UCS would be relatively small. At first sight, this line of reasoning appears to make sense. However, in evolutionary terms, and that is, after all, what preparedness is about, the distinction between auditory (noise) UCSs and tactile (shock) UCSs is not quite so convincing. As Dimond (1980) wrote:

> "Auditory functions probably owe their origins in evolution to developments from tactile or vibratory sensitivity or to general body sense mechanisms which become specialized during the course of evolution for a particular role in the analysis of the auditory signal" (p. 140).

Thus, the "belongingness" between a noise UCS and slides of snakes and spiders should not be that different from the "belongingness" between a tactile UCS and these slides. In fact, this is precisely what Cook et al. found when they combined their data from several experiments:

> "Belongingness implies a special, greater association between the phobia-relevant CS and the tactile UCS (which should be signalled by greater strength of conditioning). However, in the combined analysis, noise and shock produced equally strong conditioning to phobic stimuli" (p. 204/205).

Johnsen and Hugdahl (1988), who also compared shock and noise UCSs, reached a similar conclusion.

A second objection to the "belongingness" interpretation is that Cook et al. did not obtain independent ratings of the "belongingness" between shock or noise UCSs on the one hand, and phobia-relevant CSs, on the other hand. That this might be accomplished has been shown by Hamm and Vaitl (1985). Furthermore the "belongingness" interpretation is played down by Siddle, Bond, and Friswell (1987), who found fear-

relevance effects regardless of the type of UCS employed[4]. Finally, several studies have met the criterion of "belongingness" between UCS and CS, and, nevertheless, failed to demonstrate fear-relevance effects (see Table 2). Altogether, the concept of "belongingness" does not provide a plausible explanation for the impressive number of failures to obtain fear-relevance effects. Indeed, it is one of the sad features of the "preparedness" debate that so many authorities have gratuitously accepted the "belongingness" interpretation (e.g., Eysenck, 1987b; Marks, 1987; Öhman et al., 1985). It should be added that the present discussion does not intend to challenge the concept of "belongingness" per se. Eye-blink studies have provided substantial confirmation for the idea that the appropriateness of the CS-UCS contingencies affect conditioning (Grant, 1973). What is emphasized here, however, is that the "belongingness" concept is useless when an attempt is made to account for the varying outcomes in preparedness research.

A factor that is more likely to be involved in the question whether fear-relevance effects occur or not, is UCS intensity. It is curious that most studies failed to specify the mean UCS intensity employed. It is a well-established fact that when UCSs are intensive, responses conditioned to neutral CSs become resistant to extinction (e.g., Cambell et al., 1964), whereas in the case of mild UCSs, a stable conditioned response may not be acquired at all. Consequently, it is conceivable that there is a critical range of UCS intensities within which differential extinction of responses conditioned to phobia-relevant and neutral CSs occurs: Above or below this critical range, the experimental outcome would be very strong or weak conditioning respectively, and in both cases no differences between responses conditioned to phobia-relevant CSs and those conditioned to neutral CSs would be obtained.

Closely related to the issue of UCS intensity is the phenomenon of conditioned UCR inhibition. As noted in section 4.7, this phenomenon is totally neglected in experimental preparedness research. Yet, the possibility exists that under some conditions (e.g., relatively high UCS

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[4]At one time, even Öhman et al. (1978b) maintained that fear-relevance effects are "not specific to electric shock, because Hodes, Öhman, and Lang (...) obtained very similar data with an aversive sound as the UCS" (p. 241).

intensity), conditioned UCR inhibition develops more readily or strongly with phobia-relevant than with neutral CS+s. As conditioned UCR inhibition reflects a diminished UCS impact, it may well be that such a differential UCR inhibition is responsible for the absence of fear-relevance effects.

We may now turn to the alleged non-cognitivity of fear-relevance effects. It should be noted that, here too, replication of the results reported by Öhman (section 3.4) has proven to be very difficult, if not impossible. Thus, in spite of the fact that Cook et al. (1986; Exp. 6), Dawson et al. (1986), and Pitman and Orr (1986) all reported a fear-relevance effect in non-informed subjects, they failed to find a resistance to extinction of responses conditioned to phobia-relevant CS+s in subjects who had been informed prior to extinction that no more shocks would be delivered. McNally (1981), Eifert and Schermelleh (1985), and the study presented in section 4.6 tried to find evidence for the non-cognitivity of fear-relevance effects along different lines. McNally (1981) used an instructional reversal procedure, Eifert and Schermelleh (1985) gave their subjects language conditioning trials during extinction, and in the study presented earlier (section 4.6) subjects were given different instructions as to the contingency between CS and shock UCS. Different though these experimental paradigms may be, the authors all agree that the effect of verbal instructions on SCR extinction is not different for phobia-relevant CSs as compared to neutral CSs. All in all, the aforementioned studies failed to substantiate the non-cognitive nature of fear-relevance effects and, consequently, cast doubts on the claim (e.g., Hugdahl, 1978) that experimentally induced fear-relevance effects form an analogue to the irrationality of phobic fears.

For the sake of clarity it may be useful to summarize the critical points raised above as follows: In the past decade, the theoretical puzzles and experimental failures associated with the fear-relevance effect have multiplied at a faster rate than explanatory and empirical successes.

CHAPTER 7

CONCLUSION

As was noted earlier, the preparedness hypothesis can best be viewed as an attempt to maximize the explanatory power of the conditioning model of phobias. Having reviewed the theoretical and empirical basis of the preparedness hypothesis, we are now in a position to answer the question how successful this attempt has been. But before this is done, one point deserves special attention. In chapter 3 reference was made to the absence of an **experimentum crucis** for testing the basic tenets of the preparedness hypothesis. This does not imply that the preparedness hypothesis should be abandoned simply because it refers to an unobservable process, **in casu**, the evolutionary transmission of fear. It does imply, however, that the indirect support for this hypothesis should be scrutinized very carefully. More specifically, an hypothesis that relies on an empirically inaccessible assumption can only be accepted if it generates a set of predictions that meet two criteria. For one thing, these predictions must be corroborated by research. And in the second place, these predictions should not flow from alternative hypotheses. Thus, when the results of experimental preparedness research are evaluated, particular weight should be attached to indirect evidence that is not anticipated by more parsimonious explanations. Hence, the important question becomes: Is there any such evidence?

7.1 What is wrong with the general-process view?

If all kinds of theoretical flaws and loose ends are ignored for a moment, and it is assumed that Öhman and Seligman's versions of the preparedness hypothesis (sections 2.1, 2.2, 2.3) can be combined and treated as one coherent theoretical position, seven predictions stemming from this position can be outlined. These predictions are:

1. The selectivity of fears can be interpreted in terms of the "phylogenetic dangerousness" ("preparedness") of the objects and situations feared most.
2. The so-called "prepared" fears are easily acquired.

3. They are resistant to extinction.
4. They are insensitive to instructions.
5. Animal phobias have an earlier age of onset than social phobias.
6. Animal phobias are characterized by a higher amount of sympathetic reactivity, compared with social phobias.
7. Animal phobias are characterized by a higher amount of automatic processing, compared with social phobias.

Table 1 shows to what extent research has borne out these predictions. A cursory glance at this table reveals that question marks and vacant entries dominate the state of the art in preparedness research.

Fear survey studies have shown that "preparedness" is not the only or even the most likely factor underlying the selectivity of fears (section 4.2). Additionally, the fear survey study by Kirkpatrick (1984) reported a fear hierarchy that is, in some respects, difficult to reconcile with the preparedness hypothesis (section 6.1).

Clinical studies have yielded mixed results: Some researchers found "prepared" fears to make up the majority of the clinical fears (e.g., De Silva et al., 1977). There is, however, one study that failed to replicate this finding (section 4.3). Similarly, it is far from clear whether "preparedness" is related to treatment outcome or not. The clinical studies agree in that none of them found a relationship between "preparedness" and ease of acquisition (defined by early or sudden onset). Finally, there is evidence that animal phobias are associated with an earlier age of onset than social phobias (Öhman et al., 1985). Meanwhile, Lader's research (1980) does not seem to correspond with the assumption that animal phobias are characterized more by sympathetic reactivity than social phobias are.

The illusory correlation experiments have documented that humans tend to relate negative outcomes to stimuli such as snakes. Yet, it is by no means sure to what extent this phenomenon is based on the "phylogenetic dangerousness" rather than on the salience of these stimuli (section 6.3). Appropriate control stimuli were lacking in the illusory correlation studies.

Table 1. PREDICTIONS AND RESULTS OF PREPAREDNESS RESEARCH.

Predictions	Fear Surveys	Clinical Studies	Illusory Bias Paradigm	Conditioning Studies	Animal Studies
Selectivity of fears due to "preparedness"	-	?	?	?	+
Easy acquisition of "prepared" fears		-		-	
Resistance to extinction of "prepared" fears		?		?	
Insensitivity to instructions of "prepared" fears				?	
Differences in age of onset		+			
Differences in sympathetic activity		?			
Differences in automatic processing					

A minus sign (-) indicates that, on the whole, research has yielded evidence that does not support the predictions. A plus sign (+) refers to confirmative evidence. A question mark (?) indicates that research has yielded mixed or inconclusive results. The absence of a sign indicates that no relevant research data are available.

The psychophysiological studies are a morass of conflicting results: If we skip over the theoretical and interpretative problems, Öhman's studies seem to indicate that the extinction curve of conditioned responses is selectively affected by "phylogenetically" but not by "ontogenetically dangerous" or neutral stimuli. Furthermore, this fear-relevance effect is allegedly not modulated by instructions (i.e., is of a non-cognitive nature). Yet, fear-relevance effects only rarely occur during acquisition (section 3.4). Other studies either failed to replicate the differential extinction curves (e.g., section 4.5, 4.6) or found a retarded extinction for "ontogenetically dangerous" stimuli (Johnsen & Hugdahl, 1988). The non-cognitivity of fear-relevance effects was not corroborated in subsequent research (section 6.6).

The animal studies have provided quite solid evidence that monkeys without any previous experience (laboratory reared monkeys) do acquire selective conditioned fear responses: When confronted with models who react fearfully to snake and flower stimuli, laboratory reared monkeys acquire a phobia of the former but not of the latter category of stimuli (section 3.5).

In sum, from an empirical point of view the preparedness hypothesis is in bad shape. Even the positive experimental evidence is far from persuasive. The different ages of onset of animal and social phobias may originate from developmental rather than evolutionary factors. That is to say, it is likely that animal stimuli become salient in an earlier developmental phase than unfamiliar social situations. Admittedly, such a developmental perspective is an essential part of the line of argument followed by Öhman et al. (1985). However, on the basis of the evidence that is available, there is no need to postulate that this developmental perspective has to be extended with evolutionary notions.

As for the animal studies, it must be concluded that they have a limited significance exactly because the results are obtained with monkeys. This is not to assert that animal studies cannot be an important source of knowledge. The animal studies by Mineka (1987) fit in nicely with the steadily growing number of experiments that document biases in animal learning. To date, no experimental psychologist can reasonably argue that evolutionary boundaries do not exist in animal conditioning.

Together with phenomena such as taste aversion (Garcia & Koelling, 1965) and autoshaping of the key peck response (Brown & Jenkins, 1968), Mineka's studies clearly point out the important role of prepared associations in the classical and operant conditioning of rats, monkeys, and pigeons. The preparedness hypothesis of phobias is, however, about a different species with a different ecological niche. Seligman (1971) asserts that phobias "comprise a relatively nonarbitrary and limited set of objects: agoraphobia, fear of specific animals, insect phobias, fear of heights and fear of the dark, etc. (...) And only rarely, if ever, do we have pajama phobias, grass phobias, electric-outlet phobias, hammer phobias" (p. 112). This statement forms the preliminary to his claim that prepared biases also operate in the etiology of human fears. However, Seligman's statement is not only an unproven claim, but it is also tendentious in that it ignores the fact that humans **en masse** travel by aeroplanes, domesticate wild animals, visit dark cinemas, drive in fast cars, briefly, perform numerous acts that incorporate an element that might be construed as "dangerous in an evolutionary sense".

So, for the moment, there is no compelling reason to abandon a general-process view of learning as far as the etiology of adult fears is concerned. Schwartz (1974) rightly remarked that

> "The capacity for and frequency of arbitrary learning may increase with increasing species complexity and developmental progression. It seems clear that most of the learning done by human adults is arbitrary, or at least largely independent of evolutionary constraints" (p. 195).

7.2 A future for conditioning models of phobias?

One's evaluation of the failure of the preparedness hypothesis to offer a revised conditioning model that is both clinically satisfactory and empirically promising largely depends on the point of view that is adopted. From a clinical point of view, the lack of success of the preparedness hypothesis may strengthen the idea that a learning approach to the etiology of phobias is doomed to fail, and that cognitive theories will provide a more plausible explanation (e.g., Mackay, 1983).

From an experimental point of view, it could be argued that the crucial mistake made by the advocates of the preparedness hypothesis is their attempt to fill the black box with untestable assumptions. The lesson to be learned from this might be that, in order to gain insight into the black box, more directly observable events should be studied. Eventually, a strategy like this might provide us with an ideal conditioning model of phobias. In fact, this is the strategy defended by Maltzman (1987) when he writes that

> "The most exciting and fundamental relevant developments are occurring in the neurosciences overlapping with psychology. Investigators of human conditioning should be seeking points of contact with developments there, not with computer metaphors or folk psychology" (p. 145).

How realistic is the approach recommended by Maltzman when the etiology of human phobias has to be accounted for? An answer can be given after the following points have been considered:

1. Is there any evidence for the assumption that human phobias are based on a conditioning process? That is to say, do phobias result from CS-UCS associations?
2. If so, is it, in principle, possible to develop a provisional laboratory analogue of the etiology of phobias?
3. If so, are there, indeed, neuroscientific findings that can take this provisional model some steps further?

As for the first question, several retrospective studies examined the origins of phobias in some detail. Most of these studies have followed Rachman's (1978) classificatory scheme. This scheme describes three pathways to fear. The first pathway is classical conditioning or, in other words, the confrontation with an aversive UCS (i.e., "conditioning experiences", Öst & Hugdahl, 1981). The second pathway is observational learning (i.e., vicarious fear acquisition) of the type studied by, for example, Mineka (1987). The third pathway is learning by means of the transmission of negative information.

In a study based on analog snake phobics (N=177), Murray and Foote (1979) reported that observational learning and instructional experiences

accounted for a large majority of the fears. Their finding is at variance with an earlier study by Rimm, Janda, Lancaster, Nahe, and Dittmar (1977). Also working with analog phobics (N=45), these authors found that 36% had had aversive conditioning experiences, while observational and instructional experiences were reported less frequently, the percentages being 6% and 9%, respectively. Comparable results were reported by McNally and Steketee (1985) and by Hekmat (1987).

It is important to note that most of the above-cited studies concentrated on relatively mild, analog fears. The studies by Öst (1985, 1989) have provided solid evidence that when severe, clinical phobias (i.e., animal, social, blood, dental, claustro, and agoraphobias) are examined, conditioning experiences account for a small majority of all fears. Using a reliable, specially designed questionnaire concerning the origins of phobias, Öst repeatedly documented that some 60% of all patients remember direct, aversive experiences of the conditioning type. For example, in their sample (N=110) Öst and Hugdahl (1981) found that 58% of the patients ascribed their phobias to conditioning experiences, 17% ascribed their phobias to vicarious learning, and 10% ascribed their phobias to instructional learning. In a subsequent study (N=183), Öst (1985) reported remarkably similar percentages: 65%, 14%, and 7% for conditioning, vicarious learning, and instructional learning respectively. These findings were, again, replicated by Öst (1989) in a study based on 370 patients. In this study, separate analyses were carried out for each diagnostic subgroup. The conditioning pathway to fear was found to preponderate in all subgroups, but was especially pronounced in agoraphobia (81%) and least dominant in blood (45%) and animal (48%) phobias. Consequently, the popular view that aversive conditioning experiences are rare among phobics (e.g., Lazarus, 1971) can be rejected as being simply untrue.

The second question can now be focussed on. The findings presented above strongly suggest that it makes sense to think of phobias in terms of a CS-UCS association. Yet, laboratory models of the type proposed by Geer (1968), Deitz (1982), or Öhman et al. (1978a) are inadequate for a number of technical reasons. In the first place, these models mainly consist of multiple CS-UCS acquisition trials and, as a result, take a multiple event causation of phobias for granted. But given the fact that

a single event causation is far more common among phobias than a multiple event causation (Wolpe, 1981), a laboratory model that is based on one-trial conditioning seems to provide a better starting-point.

In the second place, a conditioning procedure consisting of multiple CS-UCS associations will result in the development of both a conditioned response and a conditioned inhibition of the unconditioned response (section 4.7). That is, responses to the CS will increase in magnitude whereas responses to the UCS will decrease. Öhman et al. (1978a) and Deitz (1982) maintain that the conditioned response is a laboratory equivalent of phobic fear. These investigators fail to pay attention to the phenomenon of conditioned inhibition. The reason for this is clear: Within the context of phobias, it is not easy to conceive of a clinical parallel of conditioned inhibition. At the same time, it is relatively arbitrary to regard conditioned responses as the most important result of a multiple CS-UCS procedure merely because these responses are, in some respects, akin to phobic reactions (Merckelbach & de Jong, 1988). Bearing in mind that multiple CS-UCS associations also produce conditioned inhibition, it might as well be argued that this procedure is a model of the reduced impact of predictable UCSs (e.g., Badia, Suter & Lewis, 1967). As a successful one-trial conditioning procedure only results in a conditioned response, it avoids the problem whether conditioned responses or conditioned inhibition should be the primary focus of attention.

In the third place, while avoidance responses constitute a crucial element of phobic behavior, human studies examining the parallels between aversive Pavlovian procedures and phobias have rarely concentrated on the conditioning of these responses. Using a classical conditioning approach, Malloy and Levis (1988) recently showed that stable avoidance responses can be elicited in healthy subjects, especially when these subjects are confronted with serial CSs. Indeed, the study by Malloy and Levis demonstrated that on the average, some 200 trials are needed before a reliable extinction of avoidance responses occurs.

A final point to be noted is that the conditioning models proposed by Öhman et al. (1978a) and Deitz (1982) are largely based on a contiguity interpretation of classical conditioning. These models emphasize that it is the temporal contiguity or coincidence of CS and UCS that forms the critical force behind the occurrence of a conditioned response.

The contiguity interpretation is undoubtedly still very popular among behavior therapists. Yet, as Reiss (1980) and Eelen (1982) convincingly demonstrated, contiguity is, at best, only one part of the story. Subjects' expectations regarding the occurrence of a UCS (Reiss, 1980) and contingency between CS and UCS (Eelen, 1982) play an equally important part in the dynamics of conditioning. To admit this is to accept a predictiveness view of classical conditioning (Rescorla & Wagner, 1972; McSweeney & Bierley, 1984). This view purports that mere temporal coincidence of CS and UCS is not sufficient for a conditioned response to occur. Except for coincidence, the UCS must be unexpected (i.e., can not be predicted on the basis of previous CSs), so that organisms are motivated to search for new CSs that can predict the occurrence of the UCS better. Rescorla (1968), for example, showed that when UCS-alone trials are interspersed among CS-UCS contiguity trials, the amount of conditioning to the CS decreases: Although contiguity is present in this procedure, the UCS-alone trials reduce the predictive value of the CS.

Conditioning phenomena such as latent inhibition (Siddle & Remington, 1987), blocking (Kamin, 1969), UCS pre-exposure effects (Randich & Lolordo, 1979), UCS re-evaluation effects (Davey, 1987) can only be properly understood from a predictiveness perspective on classical conditioning. To take just one example, a latent inhibition procedure in which a CS is presented several times without UCS undermines conditioning effects of subsequent CS-UCS associations. Although this procedure meets the contiguity criterion, its detrimental effect on conditioning can best be explained if it is assumed that CS-alone trials reduce the predictive value of the CS. While conditioning phenomena such as latent inhibition and blocking can potentially illuminate why some CS-UCS associations result in conditioned responses whereas others do not, Öhman et al. (1978) and Deitz (1982) have failed to take these phenomena into account.

In sum, a single-trial conditioning procedure in which blocking, latent inhibition, and so on are systematically examined and in which also indices of avoidance are monitored may, in due course, yield a provisional model of phobias. One final point that remains to be discussed is the extent to which this provisional model could benefit from a neuroscientific approach. This section will conclude with two examples which very clearly illustrate that a better understanding of

aversive conditioning can be obtained by studying the biological hardware in the black box.

By now it has become a well-established fact that hormonal levels play a critical part in the effects of conditioning. Indirect evidence comes from conditioning experiments in which electrodermal acquisition and extinction curves of premenstrual and intermenstrual women were compared with each other (e.g., Vila & Beech, 1977; van der Molen, Merckelbach & van den Hout, 1988). Although the effects are not dramatic, these studies reported findings suggesting an enhanced acquisition of conditioned SCRs in premenstrual women. Investigations of the way in which hormonal processes affect conditionability may shed light on the skewed sex distribution of some anxiety disorders (e.g., agoraphobia; Emmelkamp, 1982).

Even more important is research directly examing the effects of neurohormones such as ACTH (adrenocorticotropine hormone) and vasopressin on conditioning (see reviews by Eysenck & Kelley, 1987; Kelley, 1987). Recent animal studies have convincingly shown that ACTH and vasopressin injections potentiate conditioning effects. That is to say, they strongly retard extinction of responses to the CS and, under some conditions, notably a short CS duration, even give rise to an incubation of responses to the CS. It is not unlikely that ACTH and vasopressin affect conditioning through their modulation of limbic activity (de Wied & Jolles, 1982). In psychological terms, the incubation mediating effects of ACTH and vasopressin may be taken to mean that these hormones have a disinhibitory influence on attention (Kelley, 1987). Indeed, human studies (e.g., Miller et al., 1974) have documented that both ACTH and vasopressin undermine electro-encephalographic (EEG) synchronization patterns in a habituation procedure.

It is likely that there is an important connection between ACTH and vasopressin on the one hand and endogenous opioids such as beta-endorphin on the other hand. There is evidence for supposing that these neurohormones have opposite effects. Animal studies have shown that beta-endorphin directly administered to the central nervous system enhances extinction of conditioned reactions (see review by Kelley, 1987). Furthermore, in human studies, extremely negative correlations have been found between anxiety and beta-endorphin plasma levels (e.g., Post,

Pickar, Ballenger, Naber & Rubinow, 1984). From these studies, Eysenck and Kelley (1987) inferred that low levels of endogenous opioids predispose to clinical anxiety.

On the basis of an extensive review of animal and human data, Kelley (1987) suggests that

> "clinical anxiety is an outcome of an interaction between associative conditioning and individual differences in the functioning of the neuroendocrine system. The model takes as an assumption that there is a synergism between these two factors and that each alone is largely insufficient to produce clinical neuroses" (p. 403).

The first attempts to mimic incubation of phobic reactions in humans by experimental manipulations involving both neurohormone administration and aversive conditioning yielded disappointing results (e.g., Miller, Fischer, Groves, Rudrauff & Kaskin, 1977). Yet, these attempts were hampered by a number of procedural difficulties (Kelley, 1987). Thus, it seems worthwhile to subject Kelley's proposition to an empirical test using more sophisticated research designs.

Studies concerned with cerebral lateralization form the second line of neuroscientific research that may extend our knowledge of aversive conditioning. Recently, a number of researchers have tried to develop a new perspective on classical conditioning, thereby using cognitive terms such as signal value (Rescorla & Wagner, 1972), expectancy (Reiss, 1980), and attribution (Eelen, 1982). This cognitive reformulation may be particularly meaningful if it is backed up by neuropsychological knowledge (Birbaumer, 1986; Hugdahl, 1988). Two neuropsychological issues might be especially relevant in this context.

First, there are reasons to assume that there is a division of emotional labour between the two cerebral hemispheres. The view that the left hemisphere is relatively more involved in positive emotions and approach, whereas the right hemisphere is relatively more involved in negative emotions and avoidance (Gainotti, 1972; Fox & Davidson, 1984; Silberman & Weingartner, 1986) seems to account for a whole range of clinical (e.g., Weber & Sackheim, 1984), electrophysiological (e.g.,

Dimond & Farrington, 1977), and behavioral (e.g., Drake, 1987; Merckelbach & van Oppen, 1989) studies that have reported cerebral asymmetries as to emotions. Admittedly, this view has not gone unchallenged (Tucker, 1981; Levy, 1983). In particular, it remains to be seen whether it holds for both emotional appraisal and emotional expression (Hirschman & Safer, 1982). Nevertheless, it might be fruitful to examine whether this neuropsychological hypothesis about emotions has something to offer. Although a systematic investigation of the relationship between conditioning and lateralization has only just begun (see review by Hugdahl, 1987), there are some older, almost forgotten, findings in the conditioning literature that become more significant when the above-mentioned neuropsychological hypothesis is taken into account. For example, using a dichotic shadowing task, Corteen and Wood (1972) reported that a verbal CS (i.e., city names) previously paired with shock, elicits a conditioned electrodermal response when it is presented in the unattended channel. Subsequent studies (e.g., Dawson & Schell, 1982) demonstrated that this phenomenon of pre-attentive conditioning occurs exclusively when the CS is initially presented to the right hemisphere (i.e., left ear). When the CS is presented to a right, unattended channel (i.e., with initial left hemisphere processing) it fails to evoke a conditioned SCR. As the discussion of this finding has largely centred around the problem of automatic, preconscious conditioning, the possible connection between this finding and cerebral asymmetries has largely remained unnoticed (see Hugdahl, 1988). Indeed, the question arises whether the right hemisphere plays a special part in sustaining aversive conditioning.

This question is closely related to the second issue; individual differences in cerebral activation. It is likely that there are relatively stable, individual differences in hemispheric reliance. One noninvasive way of assessing a person's preference for one hemisphere or the other is recording conjugate lateral eyemovements (CLEMs) that occur when a subject is confronted with reflective questions. Most studies using CLEMs as the primary independent variable assumed that subjects with frequent leftward CLEMs (left movers) have a preference for the right hemisphere, whereas subjects with frequent rightward CLEMs (right movers) have a preference for the left hemisphere. This assumption is justified by EEG (e.g., Shevrin, Smokler & Kooi, 1980) and regional cere-

bral blood flow (e.g., Gur & Reivich, 1980) studies showing that CLEMs provide a valid index of contralateral hemispheric activation. Interestingly enough, several studies reported that cardiac perception is strongly related to leftward CLEMs (Hantas, Katkin & Reed, 1984; Montgomery & Jones, 1984). Generally speaking, subjects are rather bad at detecting their own heartbeats without training. However, a notable exception to this is formed by people with a strong right hemispheric reliance, as indexed by frequent leftward CLEMs. These people display a remarkably accurate heartbeat perception. At a theoretical level, this finding has been taken to mean that there are great differences in the extent to which individuals make use of right hemispheric functions that subserve perception of autonomic activity (Katkin, 1985). Given the fact that good heartbeat perceivers have higher state anxiety scores (Schandry, 1981) and are more upset by aversive slides (Katkin, 1985) than poor heartbeat perceivers, it is not too far-fetched to hypothesize that there is a relationship between right hemispheric reliance and accurate heartbeat perception on the one hand and conditionability on the other hand. A study by Hugdahl, Franzon, Andersson, and Walldebo (1983) lends further support to this hypothesized relationship. These investigators found that stimuli presented tachistoscopically only elicit a cardiac acceleration when they are presented in the left visual field (i.e., to the right hemisphere).

To the present author's knowledge, the possible relationship between right hemispheric reliance and conditionability has never been subjected to direct empirical tests. Here, then, is a further opportunity to update the conditioning model of phobias by neuroscientific investigations.

7.3 Epilogue

In a case study entitled "From the history of an infantile neurosis", Freud (1914, 1979) remarked:

> "I fully agree with Jung in recognizing the existence of this phylogenetic heritage; but I regard it as a methodological error to seize on a phylogenetic explanation before the ontogenetic possibilities have been exhausted" (p. 337).

Obviously, these words must be appreciated against the background of the rivalry and struggle for power within the psychoanalytical movement. Yet, Freud's remark contains an essential truth that applies equally well to the preparedness hypothesis. This hypothesis was invoked to account for a number of problems that the traditional conditioning model of phobias was facing. Its solution mainly consisted of several speculative assumptions regarding the phylogenetic origins of phobias. These assumptions are, by their very nature, empirically inaccessible. The indirect evidence that should support these assumptions is, by all standards, meager. Most importantly, however, the preparedness hypothesis is premature and has been so right from the beginning.

Well begun is half done. But the preparedness hypothesis started with a complete disregard of more parsimonious, ontogenetic explanations. When the shortcomings of the conditioning model of phobias (section 1.1) are reconsidered now, it is evident that there are a number of ontogenetic solutions that might eliminate these shortcomings.

First, the selectivity of fears may simply be accounted for in terms of well-documented conditioning phenomena such as latent inhibition and overshadowing: Some stimuli are unfamiliar and (or) salient and, consequently, are easily associated with fear.

Second, the fact that some UCSs (e.g., air raids) fail to elicit phobic fears may be understood when it is taken into account that a CS has to be predictive in order to become effective and that UCS re-evaluations (UCS - alone presentations) undermine conditioning effects. That is, when the contingency between a CS (e.g., siren) and UCS (e.g., bombs) is poor, conditioning will not take place.

Third, although not all phobias are initiated by a confrontation with a UCS, a small majority of them is. There is sufficient evidence to justify this statement (Öst, 1989). A scientific analysis of the etiology of phobias should take this fact as its point of departure. In time, it may become clear that observational learning and learning through negative information transmission is not very different from classical conditioning (e.g., Mineka, 1987; Eifert, 1987).

Fourth, in the conditioning literature, some incidental examples of single-trial conditioning can be found that seem to provide an excellent simulation of the etiology of phobias. Campbell et al. (1964), for

example, showed that, even after a considerable period of time, subjects react intensely to a tone CS if they had once experienced an association between this tone CS and respiratory paralysis induced by scoline. Nevertheless, Eysenck (1979) is probably right when he suggests that one-trial conditioning often fails to elicit stable conditioned responses. However, the question is not: Does one-trial conditioning generally work? Instead, the appropriate question is: In whom does one-trial conditioning result in a stable or incubating conditioned response?

Fifth, the skewed sex distribution of phobias may be elucidated when the interaction of hormonal processes and conditioning is studied more closely. The different onset ages of phobias is possibly related to the successive developmental phases in which various potentially phobic cues become salient. Finally, it may well be that in some persons fear is incubated rather than extinguished because of neurohormonal or neuro-psychological factors.

In fine, it would be surprising, if not suspect, to find the preparedness hypothesis surviving the 1990s.

REFERENCES

Agras, W.S., Sylvester, D. & Oliveau, D. (1969), The epidemiology of common fears and phobias. **Comprehensive Psychiatry, 10**, 151-156.

Badia, P., Suter, S. & Lewis, P. (1967), Preference for warned shock: Information and/or preparation. **Psychological Reports, 20**, 271-274.

Barry, R.J. & Maltzman, I. (1985), Heart rate deceleration is not an orienting reflex; Heart rate acceleration is not a defensive reflex. **Pavlovian Journal of Biological Science, 20**, 15-28.

Beck, A.T., Emery, G. & Greenberg, R.L. (1985), **Anxiety Disorders and Phobias: A Cognitive Perspective**. New York, Basic Books.

Benedict, B. (1979), The dangers of analogy in human ethology. **Behavioral and Brain Sciences, 2**, 1-57.

Bennet-Levy, J. & Marteau, T. (1984), Fear of animals: What is prepared? **British Journal of Psychology, 75**, 37-42.

Berghe, van den P.L. (1982), Resistance to biological self-understanding. **Behavioral and Brain Sciences, 5**, 1-37.

Birbaumer, N. (1986), Behavior therapy and brain research: A happy marriage. In J. Vinck, W. Vandereycken, O. Fontaine & P. Eelen (Eds.), **Topics in Behavioral Medicine**, Lisse, Swets, pp. 9-30.

Björkstrand, P.A. (1987), Conditioned stimulus pre-exposure and latent inhibition in human electrodermal conditioning: Effects of fear-relevant and fear-irrelevant stimuli (Abstract). **Psychophysiology, 24**, 580.

Blest, A.D. (1957), Function of eyespot patterns in the Lepidoptera. **Behavior, 11**, 210-254.

Bolles, R.C. (1971), Species-specific defense reactions. In F.R. Brush (Ed.), **Aversive Conditioning and Learning**, New York, Academic Press, pp. 72-101.

Bregman, E.O. (1934), An attempt to modify the emotional attitudes of infants by the conditioned response technique. **Journal of Genetic Psychology, 45**, 169-198.

Breland, K. & Breland, M. (1961), The misbehavior of organisms. **American Psychologist, 16**, 681-684.

Brown, P.L. & Jenkins, H.M. (1968), Auto-shaping of the pigeon's key-peck. **Journal of the Experimental Analysis of Behavior, 11**, 1-8.

Campbell, D., Sanderson, R.E. & Laverty, S.G. (1964), Characteristics of a conditioned response in human subjects during extinction trials following a single traumatic conditioning trial. **Journal of Abnormal and Social Psychology, 68**, 627-639.

Cirlot, J.E. (1962), **A Dictionary of Symbols**. London, Routledge and Kegan Paul.

Cook, E.W., Hodes, R.L. & Lang, P.J. (1986), Preparedness and phobia: Effects of stimulus content on human visceral conditioning. **Journal of Abnormal Psychology, 95**, 195-207.

Cook, M. & Mineka, S. (1987), Second order conditioning and overshadowing in the observational conditioning of fear in monkeys. **Behaviour Research and Therapy, 25**, 349-364.

Cornelius, R.R. & Averill, J.R. (1983), Sex differences in fear of spiders. **Journal of Personality and Social Psychology, 45**, 377-383.

Corteen, R.S. & Wood, B. (1972), Autonomic response to shock-associated words in an unattended channel. **Journal of Experimental Psychology, 94**, 303-318.

Costello, C.G. (1982), Fears and phobias in women: A community study. **Journal of Abnormal Psychology, 91**, 280-286.

Darwin, C. (1872, 1965), **The Expression of the Emotions in Man and Animals**. Chicago, University of Chicago Press.

Davey, G.C.L. (1987), An integration of human and animal models of Pavlovian conditioning: Associations, cognitions, and attributions. In G.C.L. Davey (Ed.). **Cognitive Processes and Pavlovian Conditioning in Humans**. Chichester, Wiley, pp. 83-114.

Dawson, M.E. & Furedy, J.J. (1976), The role of awareness in human differential autonomic classical conditioning: The necessary-gate hypothesis. **Psychophysiology, 13**, 50-53.

Dawson, M.E. & Schell, A.M. (1982), Electrodermal responses to attended and nonattended significant stimuli during dichotic listening. **Journals of Experimental Psychology, 104**, 315-325.

Dawson, M.E. & Schell, A.M. (1987), Human autonomic and skeletal classical conditioning: The role of conscious cognitive factors. In G.C.L. Davey (Ed.). **Cognitive Processes and Pavlovian Conditioning in Humans**. Chichester, Wiley, pp. 27-57.

Dawson, M.E., Schell, A.M. & Tweddle Banis, H.T. (1986), Greater resistance to extinction of electrodermal responses conditioned to potentially phobic CSs: A noncognitive process? **Psychophysiology**, 23, 522-561.

De Horne, D.J. (1980), Behaviour therapy and the treatment of anxiety. In G.D. Burrows & B. Davies (Eds.). **Handbook of Studies on Anxiety**. Amsterdam, Elsevier/North-Holland, pp. 231-264.

Deitz, S.R. (1982), Individual differences in electrodermal response conditioning and self-report of discomfort: A phobia analogue. **Physiological Psychology**, 21, 239-245.

Delprato, D.J. (1980), Hereditary determinants of fears and phobias: A critical review. **Behavior Therapy**, 11, 79-103.

Delprato, D.J. (1987), Developmental interactionism: An integrative framework for behavior therapy. **Advances in Behaviour Research and Therapy**, 9, 173-205.

Dember, W.N. (1974), Motivation and the cognitive revolution. **American Psychologist**, 29, 161-168.

De Silva, P., Rachman, S. & Seligman, M.E.P. (1977), Prepared phobias and obsessions: Therapeutic outcome. **Behaviour Research and Therapy**, 15, 65-77.

De Silva, P. (1988), Phobias and preparedness: Replication and extension. **Behaviour Research and Therapy**, 26, 97-98.

Dimberg, U. (1982), Facial reactions to facial expressions. **Psychophysiology**, 19, 643-647.

Dimberg, U. (1986a), Facial expressions as excitatory and inhibitory stimuli for conditioned autonomic responses. **Biological Psychology**, 22, 37-57.

Dimberg, U. (1986b), Facial reactions to fear-relevant and fear-irrelevant stimuli. **Biological Psychology**, 23, 153-161.

Dimberg, U. (1987), Facial reactions, autonomic activity and experienced emotions: A three component model of emotional conditioning. **Biological Psychology**, 24, 105-122.

Dimberg, U., Fredrikson, M. & Lundquist, O. (1986), Autonomic reactions to social and neutral stimuli in subjects high and low in public speaking fear. **Biological Psychology**, 23, 223-233.

Dimberg, U. & Öhman, A. (1983), The effects of directional facial cues on electrodermal conditioning to facial stimuli. **Psychophysiology**, **20**, 160-167.

Dimond, S.J. (1980), **Neuropsychology: A Textbook of Systems and Psychological Functions of the Human Brain**. London, Butterworth.

Dimond, S.J. & Farrington, L. (1977), Emotional responses to films shown to the right of left hemisphere of the brain measured by heart rate. **Acta Psychologica**, **41**, 255-260.

Donegan, N.H. & Wagner, A.R. (1987), Conditioned diminution and facilitation of the UR: A sometimes opponent-process interpretation. In I. Gormezano, W.F. Prokasy & J.K. Thompson (Eds.) **Classical Conditioning. Revised Edition**, Hillsdale NJ, Erlbaum, pp. 339-369.

Drake, R.A. (1987), Effects of gaze manipulation on aesthetic judgments: Hemisphere priming of affect. **Acta Psychologica**, **65**, 91-99.

Edelberg, R. (1973), Mechanisms of electrodermal adaptations for locomotion, manipulation, or defense. In E. Stellar & J.M. Sprague (Eds.), **Progress in Physiological Psychology. Vol** 5. New York, Academic Press, pp. 155-209.

Eelen, P. (1982), Conditioning and attribution. In J. Boulougouris (Ed.). **Learning Theory Approaches to Psychiatry**. New York, Wiley, pp. 4-17.

Eibl-Eibesfeld, I. (1979), Human ethology: Concepts and implications for the sciences of man. **Behavioral and Brain Sciences**, 2, 1-57.

Eifert, G.H. (1987), Language conditioning: Clinical issues and applications in behavior therapy. In H.J. Eysenck & I. Martin (Eds.), **Theoretical Foundations of Behavior Therapy**. New York, Plenum Press, 167-193.

Eifert, G.H. & Schermelleh, K. (1985), Language conditioning, emotional instructions, and cognitions in conditioned responses to fear-relevant and fear-irrelevant stimuli. **Journal of Behavior Therapy and Experimental Psychiatry**, **16**, 101-109.

Ekman, P. (1973), Cross-cultural studies of facial expressions. In P. Ekman (Ed.), **Darwin and Facial expressions**. New York, Academic Press, pp. 4-25.

Ekman, P. & Friesen, W.V. (1975), **Unmasking the Face**. Englewood Cliffs NJ, Prentice-Hall.

Ellsworth, D.C. (1975), Direct gaze as a stimulus for aggression. In P. Pliner, L. Kramer & T. Alloway (Eds.). **Nonverbal Communication of Aggression**. New York, Plenum Press, pp. 53-76.

Emerson, E. & Lucas, H. (1981), "Preparedness" and the development of aversive associations. **British Journal of Clinical Psychology**, 20, 293-294.

Emmelkamp, P.M.G. (1982), **Phobic and Obsessive Compulsive Disorders: Theory, Research and Practice**. New York, Plenum Press.

English, H.B. (1929), Three cases of the "conditioned fear response". **Journal of Abnormal and Social Psychology**, 34, 221-225.

Eysenck, H.J. (1968), A theory of the incubation of anxiety/fear responses. **Behaviour Research and Therapy**, 6, 309-321.

Eysenck, H.J. (1976), The learning theory model of neurosis: A new approach. **Behaviour Research and Therapy**, 14, 251-267.

Eysenck, H.J. (1979), The conditioning model of neurosis. **Behavioral and Brain Sciences**, 2, 155-199.

Eysenck, H.J. (1982), Neobehavioristic (S-R) theory. In G.T. Wilson & C.M. Franks (Eds.), **Contemporary Behavior Therapy**. New York, Guilford Press, pp. 205-276.

Eysenck, H.J. (1987a), Behavior therapy. In H.J. Eysenck & I. Martin (Eds.). **Theoretical Foundations of Behavior Therapy**. New York, Plenum Press, pp. 3-35.

Eysenck, H.J. (1987b), The role of heredity, environment, and "preparedness" in the genesis of neurosis. In H.J. Eysenck & I. Martin (Eds.), **Theoretical Foundations of Behavior Therapy**. New York, Plenum Press, pp. 379-402.

Eysenck, H.J. & Eysenck, S.B.G. (1964), **Manual of the Eysenck Personality Inventory**. London, University of London Press.

Eysenck, H.J. & Kelley, M.J. (1987), The interaction of neurohormones with Pavlovian A and Pavlovian B conditioning in the causation of neuroses extinction, and incubation of anxiety. In G. Davey (Ed.), **Cognitive Processes and Pavlovian Conditioning in Humans.** Chichester, Wiley, pp. 251-286.

Eysenck, H.J. & Rachman, S. (1965), **The Causes and Cures of Neurosis.** London, Routledge and Kegan Paul.

Foa, E.B. Steketee, G.S., McCarthy, P.R. McNally, R.J. & Grayson, J.B. (1988), A test of preparedness theory in anxiety-disordered patients using an operant paradigm. Submitted.

Fox, N.A. & Davidson, R.J. (1984), Hemispheric substrates of affect: A developmental model. In N.A. Fox & R.J. Davidson (Eds.), **The Psychobiology of Affective Development.** Hillsdale NJ, Erlbaum, pp. 353-381.

Fredrikson, M. (1981), Orienting and defensive reactions to phobic and conditioned fear stimuli in phobics and normals. **Psychophysiology, 18,** 456-465.

Fredrikson, M., Hugdahl, K. & Öhman, A. (1976), Electrodermal conditioning to potentially phobic stimuli in male and female subjects. **Biological Psychology, 4,** 305-314.

Fredrikson M. & Öhman, A. (1979), Cardiovascular and electrodermal responses conditioned to fear-relevant stimuli. **Psychophysiology, 16,** 1-7.

Freud, S. (1909, 1980), **Analyse der Phobie eines Fünfjährigen Knaben.** Frankfurt a/M, Fischer Verlag.

Freud, S. (1917, 1974), **Introductory Lectures on Psychoanalysis.** Harmondsworth, Penguin.

Freud, S. (1918, 1979), **From the History of Infantile Neuroses. Case Histories II.** Harmondsworth, Penguin.

Furedy, J.J. (1968), Human orienting reactions as a function of electrodermal versus plethysmographic response modes and single versus alternating stimulus series. **Journal of Experimental Psychology, 77,** 70-78.

Furedy, J.J. & Arabian, J.M. (1979), A Pavlovian psychophysiological perspective on the OR: The facts of the matter. In H.D. Kimmel, E.H. van Olst & J.F. Orlebeke (Eds.), **The Orienting Reflex in Humans**, Hillsdale NJ, Erlbaum, pp. 353-372.

Furedy, J.J. & Schiffman, K. (1971), Test of the propriety of the traditional discriminative control procedure in Pavlovian electrodermal and plethysmographic conditioning. **Journal of Experimental Psychology**, 91, 161-164.

Gainotti, G. (1972), Emotional behavior and hemispheric side of the lesion. **Cortex**, 8, 41-55.

Garcia, J., Ervin, F.R. & Koelling, R.A. (1966), Learning with prolonged delay of reinforcement. **Psychonomic Science**, 5, 121-122.

Garcia, J. & Koelling, R.A. (1966), Relation of cue to consequence in avoidance learning. **Psychonomic Science**, 4, 123-124.

Garcia, J. & Koelling, R.A. (1967), A comparison of aversions induced by X-rays, drugs, and toxins. **Radiation Research**, 7, 439-450.

Garcia, J., McGowan, B.K., Ervin, F.R. & Koelling, R. (1968), Cues-Their relative effectiveness as a function of the reinforcer. **Science**, 160, 794-795.

Garcia, J., McGowan, B.K. & Green, K.F. (1972), Biological constraints on conditioning. In A.H. Black & W.F. Prokasy (Eds.). **Classical Conditioning II. Curent Research and Theory**. New York, Appleton-Century - Crofts, pp. 3-27.

Geer, J.H. (1968), A test of the classical conditioning model of emotion: The use of nonpainful aversive stimuli as unconditioned stimuli in a conditioning procedure. **Journal of Personality and Social Psychology**, 10, 148-156.

Goorney, A.B. & O'Connor, P.J. (1971), Anxiety associated with flying. **British Journal of Psychiatry**, 119, 159-166.

Gossop, M. (1981), **Theories of Neurosis**. Berlin, Springer-Verlag.

Graham, F.K. (1973), Habituation and dishabituation of responses innervated by the autonomic nervous system. In H.V.S. Peeke & M.J. Herz (Eds.), **Habituation. Vol 1. Behavioural Studies**. New York, Academic Press, pp. 453-464.

Graham, F.K. (1978a), Normality of distributions and homogeneity of variance of heart rate and heart periode samples. **Psychophysiology**, 15, 487-491.

Graham, F.K. (1978b), Constraints on measuring heart rate and period through real and cardiac time. **Psychophysiology**, 15, 492-495.

Graham, F.K. & Clifton, R.K. (1966), Heart-rate change as a component of the orienting response. **Psychological Bulletin**, 65, 305-320.

Grant, D.A. (1973), Cognitive factors in eye-lid conditioning. **Psychophysiology**, 10, 75-81.

Gray, J.A. (1979), Is there any need for conditioning in Eysenck's conditioning model of neurosis? **Behavioral and Brain Sciences**, 2, 169-171.

Gray, J.A. (1982), **The Neuropsychology of Anxiety: An Enquiry into the Functions of the Septo-Hippocampal System**. Oxford, Clarendon.

Grayson, J.B. (1982), The elicitation and habituation of orienting and defensive responses to phobic imagery and the incremental stimulus intensity effect. **Psychophysiology**, 19, 105-111.

Grings, W.W. & Schell, A.M. (1969), Magnitude of electrodermal responses to a standard stimulus as a function of intensity and proximity of a prior stimulus. **Journal of Comparative and Physiological Psychology**, 67, 77-82.

Grings, W.W. & Schell, A.M. (1971), Effects of trace versus delay conditioning, interstimulus interval variability, and instructions on UCR diminution. **Journal of Experimental Psychology**, 90, 136-140.

Gur, R.C. & Reivich, M. (1980), Cognitive task effects on hemispheric blood flow in humans: Evidence of individual differences in hemispheric activation. **Brain and Language**, 9, 78-92.

Hailman, J.P. (1979), The ethology behind human ethology. **Behavioral and Brain Sciences**, 2, 35-36.

Hallowell, A.I. (1938), Fear and anxiety as cultural and individual variables in a primitive society. **Journal of Social Psychology**, 9, 25-47.

Hamm, A. & Vaitl, D. (1985), "Belongingness": Operationalisierung eines neuen Konzepts in der Lerntheorie. **Archiv für Psychologie**, 137, 13-27.

Hamm, A. & Vaitl, D. (1986), Kardiovaskuläre und elektrodermale Reaktionsspezifität bei essentiellen Grenzwert-Hypertonikern: Ein Konditionierungsexperiment. **Zeitschrift für Klinische Psychologie**, 3, 201-216.

Hantas, M., Katkin, E.S. & Reed, S.D. (1984), Heartbeat discrimination training and cerebral lateralization. **Psychophysiology**, 21, 274-278.

Hare, R.D. & Blevings, G. (1975a), Conditioned orienting and defensive responses. **Psychophysiology**, 12, 289-297.

Hare, R.D. & Blevings, G. (1975b), Defensive responses to phobic stimuli. **Biological Psychology**, 3, 1-13.

Harris, B. (1979), Whatever happened to little Albert? **American Psychologist**, 34, 151-160.

Hebb, D.O. (1946), On the nature of fear. **Psychological Review**, 35, 259-276.

Hebb, D.O. & Riesen, A.H. (1943), The genesis of irrelevant fears. **Bulletin of the Canadian Psychological Association**, 3, 49-50.

Heisel, S.J. (1988), An ethological analysis of obsessive-compulsive disorder. In J.R. Feierman (Ed.). **The Ethology of Psychiatric Populations**. New York, Elsevier, in press.

Hekmat, H. (1987), Origins and development of human fear reaction. **Journal of Anxiety Disorders**, 1, 197-218.

Herrnstein, R.J. (1977), The evolution of behaviorism. **American Psychologist**, 32, 593-603.

Hinde, R.A. (1974), **Biological Basis of Human Social Behavior**. New York, McGraw-Hill.

Hirschman, R.S. & Safer, M.A. (1982), Hemisphere differences in perceiving positive and negative emotions. **Cortex**, 18, 569-580.

Holland, H.C. (1974), Displacement activity as a form of abnormal behaviour in animals. In H.R. Beech (Ed.), **Obsessional States**, London, Methuen, pp. 161-173.

Hugdahl, K. (1978), Electrodermal conditioning to potentially phobic stimuli: Effects of instructed extinction. **Behaviour Research and Therapy**, 16, 315-321.

Hugdahl, K. (1987), Pavlovian conditioning and hemispheric asymmetry: A perspective. In G.C.L. Davey (Ed.). **Cognitive Processes and Pavlovian Conditioning in Humans**, Chichester, Wiley, pp. 147-182.

Hugdahl, K. (1988), Bilateral electrodermal asymmetry: Past hopes and future prospects. **International Journal of Neurosciences**, **39**, 33-34.

Hugdahl, K., Franzon, M., Andersson, B. & Walldebo, G. (1983), Heart rate responses (HRR) to lateralized visual stimuli. **Pavlovian Journal of Biological Science**, **18**, 186-198.

Hugdahl, K., Fredrikson, M. & Öhman, A. (1977), "Preparedness" and "arousability" as determinants of electrodermal conditioning. **Behaviour Research and Therapy**, 15, 345-353.

Hugdahl, K. & Kärker, A. (1981), Biological vs. experiential factors in phobic conditioning. **Behaviour Research and Therapy**, **19**, 109-115.

Hugdahl, K. & Öhman, A. (1977), Effects of instruction on acquisition and extinction of electrodermal responses to fear-relevant stimuli. **Journal of Experimental Psychology**, 3, 608-618.

Hugdahl, K. & Öhman, A. (1980), Skin conductance conditioning to potentially phobic stimuli as a function of interstimulus interval and delay versus trace paradigm. **Psychophysiology**, 17, 348-355.

Hull, C.L. (1943), **Principles of Behaviour**. New York, Appleton-Century-Crofts.

Hygge, S. & Öhman, A. (1978), Modelling processes in the acquisition of fears: Vicarious electrodermal conditioning to fear-relevant stimuli. **Journal of Personality and Social Psychology**, **36**, 271-279.

Izard, C. (1971), **The Face of Emotion**. New York, Appleton-Century-Crofts.

Jennings, J.R., Berg, W.K., Hutcheson, J.F., Obrist, P., Porges, S. & Turpin, G. (1981), Publication guidelines for heart rate studies in man. **Psychophysiology**, **18**, 226-231.

Johnsen, B.H. & Hugdahl, K. (1988), Preparedness and electrodermal fear-conditioning: An old problem revisited (abstract). **Psychophysiology**, **25**, 457-458.

Johnston, T.D. (1981), Contrasting approaches to a theory of learning. **Behavioral and Brain Sciences**, 4, 125-173.

Jones, H.E. (1930), The retention of conditioned emotional reactions in infancy. **Journal of Genetic Psychology**, 37, 485-497.

Jones, M.C. (1924), The elemination of children's fear. **Journal of Experimental Psychology**, 7, 383-390.

Jürgens, U. & Ploog, D. (1974), **Von der Ethologie zur Psychologie**, München, Kindler.

Kamin, L.J. (1969), Predictability, surprise, attention and conditioning. In B.A. Campbell & R.M. Church (Eds.), **Punishment and Aversive Behavior**. New York, Appleton-Century-Crofts, pp. 279-296.

Katkin, E.S. (1985), Blood, sweat and tears: Individual diffences in autonomic self-perception. **Psychophysiology**, 22, 125-137.

Kelley, M.J. (1987), Hormones and clinical anxiety: An imbalanced neuromodulation of attention. In H.J. Eysenck & I. Martin (Eds.), **Theoretical Foundations of Behavior Therapy**. New York, Plenum Press, pp. 403-432.

Kettlewell, H. (1973), **The Evolution of Melanism**. Oxford, Oxford University Press.

Kimble, G.A. & Ost, J.W.P. (1961), A conditioned inhibition process in eyelid conditioning. **Journal of Experimental Psychology**, **61**, 150-156.

Kimmel, H.D. (1966), Inhibition of the unconditioned response in classical conditioning. **Psychological Review**, **73**, 232-240.

Kimmel, H.D. & Pennypacker, H.S. (1962), Conditioned diminution of the unconditioned response as a function of the number of reinforcements. **Journal of Experimental Psychology**, **64**, 20-23.

Kirkpatrick, D.R. (1984), Age, gender, and patterns of common intense fears among adults. **Behaviour Research and Therapy**, **19**, 109-115.

Kitcher, P. (1985), **Vaulting Ambitions: Sociobiology and the Quest of Human Nature**. Cambridge M, MIT Press.

Kleinknecht, R.A. (1982), The origins and remission of fear in a group of tarantula enthiusiasts. **Behaviour Research and Therapy**, **20**, 437-443.

Köhler, W. (1925), **The Mentality of Apes**. New York, Harcourt.

Kroll, W. & Witte, K. (1921), **Paulys Real - Enclopädie der Classischen Altertumswissenschaft** Vol II. Stuttgart, Metzlersche Verlagsbuchhandlung.

Lader, M.H. (1969), The psychophysiology of anxiety. In M. Lader (Ed.). **Studies of Anxiety**, Kent, Headley, pp. 53-61.

Lader, M.H. (1980), Psychophysiological studies in anxiety. In J.T. Burrows & E. Davies (Eds.), **Handbook of Studies on Anxiety**, Amsterdam, Elsevier/North-Holland, pp. 59-88.

Lang, P.J. (1984), Cognition in emotion: Concept and action. In C.E. Izard, J. Kagan & R.B. Zajonc (Eds.), **Emotion, Cognition and Behavior**. Cambridge (Eng.), Cambridge University Press, pp. 192-226.

Lanzetta, J.T. & Orr, S.P. (1980), Influences of facial expressions on the classical conditioning of fear. **Journal of Personality and Social Psychology**, 39, 1081-1087.

Lanzetta, J.T. & Orr, S.P. (1981), Stimulus properties of facial expressions and their influence on the classical conditioning of fear. **Motivation and Emotion**, 5, 225-234.

Lazarus, A.A. (1971), **Behavior Therapy and Beyond**. New York, McGraw-Hill.

Levey, A.B. (1980), Measurement units in psychophysiology. In I. Martin & P.H. Venables (Eds.). **Techniques in Psychophysiology**, New York, Wiley, pp. 597-628.

Levey, A.B. & Martin, I. (1975), Classical conditioning of human "evaluative" responses. **Behaviour Research and Therapy**, 13, 221-226.

Levy, J. (1983), Individual differences in cerebral hemisphere asymmetry: Theoretical issues and experimental considerations. In J.B. Hellige (Ed.). **Cerebral Hemisphere Asymmetry: Method, Theory, Application**. New York, Praeger, pp. 110-172.

Lewis, A.J. (1934), Melancholia: A clinical survey of depressive states. **Journal of Mental Science**, 80, 277-378.

Lewontin, R.C. (1979), Sociobiology as an adaptionist program. **Behavioral Science**, 24, 5-14.

Lumsden, J., Howard, R.C. & Fenton, G.W. (1986), The contingent negative variation (CNV) to fear-related stimuli in acquisition and extinction. **International Journal of Psychophysiology**, 3, 253-261.

Lumsden, L.J. & Wilson, E.O. (1982), Précis of Genes, Mind, and Culture. **Behavioral and Brain Sciences**, 5, 1-7.

Lykken, D.T. (1968), Neuropsychology and psychophysiology in personality research. In E.F. Borgatta & W.W. Lambert (Eds.). **Handbook of Personality Theory and Research**. Chicago, Rand McNally, pp. 413-509.

Lykken, D.T., Macindoe, I. & Tellegen, A. (1972), Preception: Autonomic response to shock as a function of predictability in time and locus. **Psychophysiology**, 9, 318-333.

Lykken, D.T. & Tellegen, A. (1974), On the validity of the preception hypothesis. **Psychophysiology**, 11, 125-132.

Macdonough, T.S. (1976), Derivation of a "composite small animal" score for the fear survey schedule III. Behavioral and subjective report correlates. **Psychological Reports**, 38, 231-237.

Mackay, D. (1983), Principles of learning. In M. Weller (Ed.), The **Scientific Basis of Psychiatry**. Eastbourne, Baillière Tindall, pp. 322-333.

Malloy, P. & Levis, D.J. (1988), A laboratory demonstration of persistent human avoidance. **Behavior Therapy**, 19, 229-241.

Maltzman, I. (1979), Orienting reflexes and classical conditioning in humans. In H.D. Kimmel, E.H. van Olst & J.F. Orlebeke (Eds.). **The Orienting Reflex in Humans**. Hillsdale NJ, Erlbaum, pp. 323-351.

Maltzman, I. (1987), A neo-Pavlovian interpretation of the OR and classical conditioning in humans: With comments on alcoholism and the poverty of cognitive psychology. In G.C.L. Davey (Ed.). **Cognitive Processes and Pavlovian Contitioning in Humans**. Chichester, Wiley, pp. 221-249.

Maltzman, I. & Boyd, G. (1984), Stimulus significance and bilateral SCRs to potentially phobic pictures. **Journal of Abnormal Psychology**, 93, 41-46.

Marcel, A. (1983), Conscious and unconscious perception: Experiments on visual masking and word recognition. **Cognitive Psychology**, 15, 197-237.

Marks, I.M. (1969), **Fears and Phobias**. New York, Academic Press.

Marks, I.M. (1977), Phobias and obsessions. Clinical phenomena in search of laboratory models. In J.D. Maser & M.E.P. Seligman (Eds.). **Psychopathology: Experimental Models**. San Francisco, Freeman, pp. 174-213.

Marks, I.M. (1987), **Fears, Phobias, and Rituals. Panic, Anxiety, and their Disorders**. New York, Oxford University Press.

Marks, I.M. (1988), Advances in behaviour psychotherapy. Paper presented at the **3rd Behaviour Therapy World Congress**, Edinburgh.

Marks, I.M. & Mathews, A.M. (1979), Brief standard self-rating for phobic patients. **Behaviour Research and Therapy**, 17, 263-267.

Martin, I. & Rust, J. (1976), Habituation and the structure of the electrodermal system. **Psychophysiology**, 13, 554-562.

Martin, I. & Levey, A.B. (1978), Evaluative conditioning. **Advances in Behaviour Research and Therapy**, 1, 57-102.

May, M. (1944), **A Social Psychology of War and Peace**. New Haven, Yale University Press.

Mayr, E. (1974), Behavior programs and evolutionary strategies. **American Scientist**, 62, 650-659.

McAllister, W.R. & McAllister, D.E. (1979), Are the concepts of enhancement and preparedness necessary? **Behavioral and Brain Sciences**, 2, 177-178.

McGuire, M.T. (1986), Ethological explanations in psychiatry. Paper presented at the Sandoz Research Symposium **"Biological Measures. Their Theoretical and Diagnostic Value in Psychiatry"**, Bergen aan Zee.

McGuire, M.T. & Essock-Vitale, S.M. (1982), Psychiatric disorders in the context of evolutionary biology: The impairment of adaptive behaviors during the exacerbation and remission of psychiatric illnesses. **Journal of Nervous and Mental Disease**, 170, 9-20.

McNally, R.J. (1981), Phobias and preparedness: Instructional reversal of electrodermal conditioning to fear-relevant stimuli. **Psychological Reports**, 48, 175-180.

McNally, R.J. (1986), Pavlovian conditioning and preparedness: Effects of initial fear level. **Behaviour Research and Therapy**, **24**, 27-33.

McNally, R.J. (1987), Preparedness and phobias: A review. **Psychological Bulletin**, **101**, 283-303.

McNally, R.J. & Foa, E.B. (1986), Preparedness and resistance to extinction to fear-relevant stimuli: A failure to replicate. **Behaviour Research and Therapy**, **24**, 529-535.

McNally, R.J. & Reiss, S. (1982), The preparedness theory of phobias and human safety-signal conditioning. **Behaviour Research and Therapy**, **10**, 153-159.

McNally, R.J. & Reiss, S. (1984), The preparedness theory of phobias: The effects of initial fear level on safety signal conditioning to fear-relevant stimuli. **Psychophysiology**, **21**, 647-651.

McNally, R.J. & Steketee, G.S. (1985), The etiology and maintenance of severe animal phobias. **Behaviour Research and Therapy**, **23**, 431-435.

McNemar, A. (1969), **Psychological Statistics**, New York, Wiley.

McSweeney, F.K. & Bierley, C. (1984), Recent developments in classical conditioning, **Journal of Consumer Research**, **11**, 619-631.

Miller, L.H., Kastin, A.J., Sandman, C.A., Fink, M. & Veen, van W.J. (1974), Polypeptide influences on attention, memory, and anxiety in man. **Pharmacology, Biochemistry, and Behavior**, **2**, 663-668.

Miller, L.H., Fisher, S.C., Groves, G.A., Rudrauff, M.E. & Kaskin, A. (1977), MSH/ACTH influences on the CAR in human subjects: A negative finding. **Pharmacology, Biochemistry, and Behavior**, **7**, 417-419.

Mineka, S. (1985), Animal models of anxiety-based disorders: Their usefulness and limitations. In A.H. Tuma & J. Maser (Eds.). **Anxiety and the Anxiety Disorders**. Hillsdale NY, Erlbaum, pp. 199-244.

Mineka, S. (1987), A primate model of phobic fears. In H.J. Eysenck & I. Martin (Eds.). **Theoretical Foundations of Behavior Therapy**. New York, Plenum Press, pp. 81-111.

Mineka, S. & Kihlstrom, J.F. (1978), Unpredictable and uncontrollable events: A new perspective on experimental neurosis. **Journal of Abnormal Psychology**, **87**, 256-271.

Mineka, S. & Tomarken, A.J. (1989), The role of cognitive biases in the origins and maintenance of fear and anxiety disorders. In T. Archer & L.G. Nilsson (Eds.). **Aversion, Avoidance and Anxiety: Perspectives on Aversively Motivated Behavior**. Hillsdale NJ, Erlbaum, pp. 195-221.

Montegomery, W.A. & Jones, G.E. (1984), Laterality, emotionality, and heartbeat perception. **Psychophysiology**, **21**, 459-465.

Morrow, M.C. (1966), Recovery of conditioned UCR diminution following extinction. **Journal of Experimental Psychology**, **71**, 884-888.

Mowrer, O.H. (1939), Stimulus-response theory of anxiety. **Psychological Review**, **46**, 553-565.

Murray, E.J. & Foote, F. (1979), The origins of fear of snakes. **Behaviour Research and Therapy**, **17**, 489-493.

O'Gorman, J.G. (1977), Individual differences in habituation of human physiological responses: A review of theory, method, and findings in the study of personality correlates in non-clinical populations. **Biological Psychology**, 5, 257-318.

Öhman, A. (1979), Fear relevance, autonomic conditioning, and phobias: A laboratory model. In P.O. Sjöden, S. Bates & W.S. Dockens (Eds.). **Trends in Behavior Therapy**. New York, Academic Press, pp. 107-133.

Öhman, A. (1986), Face the beast and fear the face: Animal and social fears as prototypes for evolutionary analyses of emotion. **Psychophysiology**, **23**, 123-145.

Öhman, A. (1988), The psychophysiology of emotion: An evolutionary-cognitive perspective. In P.K. Ackles, J.R. Jennings & M.G.H. Coles (Eds.). **Advances in Psychophysiology Vol 2**, Greenwich, JAI Press, in press.

Öhman, A. & Dimberg, U. (1978), Facial expressions as conditioned stimuli for electrodermal response: A case of "preparedness"? **Journal of Personality and Social Psychology**, **11**, 1251-1258.

Öhman, A. & Dimberg, U. (1984), An evolutionary perspective on human social behavior. In W.M. Waid (Ed.). **Sociophysiology**, New York, Springer, pp. 47-86.

Öhman, A., Dimberg, U. & Öst, L.-G. (1985), Animal and social phobias: Biological constraints on learned fear responses. In S. Reiss & R.R. Bootzin (Eds.). **Theoretical Issues in Behavior Therapy**. Orlando FL, Academic Press, pp. 123-178.

Öhman, A., Esteves, F., Parra, C. & Soares, J. (1988), Brain lateralization and preattentive elicitation of conditioned skin conductance respones. (Abstract). **Psychophysiology**, 25, 473.

Öhman, A., Eriksson, A., Fredrikson, M., Hugdahl, K. & Olofsson, C. (1974), Habituation of the electrodermal orienting reaction to potentially phobic and supposedly neutral stimuli in normal human subjects. **Biological Psychology**, 2, 85-93.

Öhman, A., Eriksson, A. & Olofsson, C. (1975), One-trial learning and superior resistance to extinction of autonomic responses conditioned to potentially phobic stimuli. **Journal of Comparative and Physiological Psychology**, 88, 619-627.

Öhman, A., Erixon, G. & Löfberg, I. (1975), Phobias and preparedness: Phobic versus neutral pictures as conditioned stimuli for human autonomic responses. **Journal of Abnormal Psychology**, 84, 41-45.

Öhman, A., Fredrikson, M. & Hugdahl, K. (1978a), Towards an experimental model for simple phobic reactions. **Behavioural Analysis and Modification**, 2, 97-114.

Öhman, A., Fredrikson, M. & Hugdahl, K. (1978b), Reply to S.J. Rachman: Towards an experimental model of simple phobic reactions. **Behavioural Analysis and Modification**, 2, 240-245.

Öhman, A., Fredrikson, M. & Hugdahl, K. (1978c), Orienting and defensive responding in the electrodermal system: Palmar-dorsal differences and recovery rate during conditioning to potentially phobic stimuli. **Psychophysiology**, 15, 93-101.

Öhman, A., Fredrikson, M., Hugdahl, K. & Rimmö, P.A. (1976), The premise of equipotentiality in human classical conditioning: Conditioned electrodermal responses to potentially phobic stimuli. **Journal of Experimental Psychology**, 105, 313-337.

Öhman, A. & Hugdahl, K. (1979), Instructional control of autonomic respondents: Fear relevance as a critical factor. In N. Birbaumer & H.D. Kimmel (Eds.). **Biofeedback and Self-Regulation**. New York, Wiley, pp. 149-165.

Orr, S.P. & Lanzetta, J.T. (1980), Facial expressions of emotion as conditioned stimuli for human autonomic responses. **Journal of Personality and Social Psychology**, **38**, 278-282.

Orr, S.P. & Lanzetta, J.T. (1984), Extinction of an emotional response in the presence of facial expressions of emotion. **Motivation and Emotion**, **8**, 55-66.

Öst, L.-G. (1985), Ways of acquiring phobias and outcome of behavioral treatments. **Behaviour Research and Therapy**, **23**, 683-689.

Öst, L.-G. (1989), Age of onset in different phobias. **Journal of Abnormal Psychology**, in press.

Öst, L.-G. & Hugdahl, K. (1981), Acquisition of phobias and anxiety response patterns in clinical patients. **Behaviour Research and Therapy**, **19**, 439-447.

Pavlov, I.P. (1928), **Lectures on Conditioned Reflexes**. New York, International Publishers.

Pendery, M. & Maltzman, I. (1977), Instructions and the orienting reflex in semantic conditioning of the galvanic skin response in a innocuous situation. **Journal of Experimental Psychology**, **106**, 120-140.

Pitman, R.K. & Orr, S.P. (1986), Test of the conditioning model of neurosis: Differential aversive conditioning of angry and neutral facial expressions in anxiety disorder patients. **Journal of Abnormal Psychology**, **95**, 208-213.

Post, R.M., Pickar, D., Ballenger, J.C. Naber, D. & Rubinow, D.R. (1984), Endogenous opiates in cerebrospinal fluid: Relationship to mood and anxiety. In R.M. Post & J.C. Ballenger (Eds.), **Neurobiology of Mood Disorders**, London, Williams and Wilkens, pp. 356-368.

Prigatano, G.P. & Johnson, H.J. (1974), Autonomic nervous system changes associated with a spider phobic reaction. **Journal of Abnormal Psychology**, **83**, 169-177.

Prokasy, W.F. & Kumpfer, K.L. (1973), Classical conditioning. In W.F. Prokasy & D.C. Raskin (Eds.), **Electrodermal Activity in Psychological Research**. New York, Academic Press, pp. 157-196.

Rachman, S. (1976), The passing of the two-stage theory of fear and avoidance: Fresh possibilities. **Behaviour Research and Therapy**, **14**, 125-131.

Rachman, S. (1977), The conditioning theory of fear-acquisition: A critical examination. **Behaviour Research and Therapy**, 15, 375-387.

Rachman, S. (1978), Biologically significant fears. **Behavioural Analysis and Modification**, 2, 234-239.

Rachman, S. (1978), **Fear and Courage**, New York, Freeman.

Rachman, S. & Hodgson, R.J. (1980), **Obsessions and Compulsions**. Englewood Cliffs NJ, Prentice-Hall.

Rachman, S. & Lopatka, C. (1986), Match and mismatch in the prediction of fear-I. **Behaviour Research and Therapy**, **24**, 387-393.

Rachman, S. & Seligman, M.E.P. (1976), Unprepared phobias: "Be prepared". **Behaviour Research and Therapy**, **14**, 333-338.

Randich, A. & Lolordo, V.M. (1979), Associative and non-associative theories of the UCS pre-exposure phenomenon: Implications for Pavlovian conditioning. **Psychological Bulletin**, **86**, 523-548.

Razran, G. (1971), **Mind in Evolution**, Boston, Houghton Mifflin.

Redican, W.K. (1975), Facial expressions in nonhuman primates. In L. Rosenblun (Ed.). **Primate Behavior**. New York, Academic Press, pp. 103-194.

Reiss, S. (1980), Pavlovian conditioning and human fear: An expectancy model. **Behavior Therapy**, 11, 380-396.

Rescorla, R.A. (1968), Probability of shock in the presence and absence of the CS in fear conditioning. **Journal of Comparative and Physiological Psychology**, 66, 1-5.

Rescorla, R.A. & Wagner, A.R. (1972), A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A.A. Black & W.F. Prokasy (Eds.). **Classical Conditioning II: Current Research and Theory**. New York, Appleton-Century-Crofts, pp. 64-99.

Rimm, D.C., Janda, L.H., Lancaster, D.W., Nahl, M. & Dittmar, K. (1977), An exploratory investigation of the origin and maintenance of phobias. **Behaviour Research and Therapy**, 15, 231-238.

Samelson, F. (1980), J.B. Watson's little Albert, Cyril Burt's Twins and the need for a critical science. **American Psychologist**, 35, 619-625.

Saigh, P.A. (1984), An experimental analysis of delayed post-traumatic stress. **Behaviour Research and Therapy**, 22, 679-682.

Schandry, R. (1981), Heart beat perception and emotional experience. **Psychophysiology**, 18, 483-488.

Schell, A.M. & Grings, W.W. (1971), Judgments of UCS intensity and diminution of the unconditioned GSR. **Psychophysiology**, 8, 427-432.

Schneirla, T.C. (1965), Aspects of stimulation and organisation in approach/withdrawal processes underlying vertebrate behavioral development. In D.S. Lehrman, R.A. Hinde & E. Shaw (Eds.). **Advances in the Study of Behavior**, Vol I. New York, Academic Press, pp. 2-75.

Schwartz, B. (1974), On going back to nature: A review of Seligman and Hager's Biological Boundaries of Learning. **Journal of the Experimental Analysis of Learning**, 21, 183-198.

Seligman, M.E.P. (1970), On the generality of the laws of learning. **Psychological Review**, 77, 406-418.

Seligman, M.E.P. (1971), Phobias and preparedness. **Behavior Therapy**, 2, 307-320.

Seligman, M.E.P. (1975), **Helplessness: On Depression, Development, and Death.** San Francisco, Freeman.

Seligman, M.E.P. (1988), Competing theories of panic. In S. Rachman & J.D. Maser (Eds.). **Panic. Psychological Perspectives.** Hillsdale NJ, Erlbaum, pp. 321-329.

Seligman, M.E.P., Maier, S.F. & Solomon, R.L. (1971), Unpredictable and uncontrollable aversive events. In F.R. Brush (Ed.), **Aversive Conditioning and Learning**. New York, Academic Press, pp. 347-400.

Seligman, M.E.P. & Hager, J.L. (Eds.) (1972), **Biological Boundaries of Learning**. New York, Appleton-Century-Crofts.

Seligman, M.E.P. & Yellen, A. (1987). What is a dream? **Behaviour Research and Therapy**, 25, 1-24.

Shettleworth, S.J. (1975), Reinforcement and the organization of behavior in golden hamsters: Hunger, envinronment, and food reinforcement. **Journal of Experimental Psychology**, 103, 56-87.

Shevrin, H., Smokler, I. & Kooi, K.A. (1980), An empirical link between lateral eye movements and lateralized event-related brain potentials. **Biological Psychiatry**, 15, 695-697.

Siddle, D.A.T., Bond, N.W. & Friswell, R. (1987), Effects of stimulus content on second-order electrodermal conditioning in humans. **Psychophysiology**, 24, 439-448.

Siddle, D.A.T. & Remington, B. (1987), Latent inhibition and human Pavlovian conditioning: Research and relevance. In G.C.L. Davey (Ed.). **Cognitive Processes and Pavlovian Conditioning in Humans**, Chichester, Wiley, pp. 113-146.

Siegel, S. (1956), **Nonparametric Statistics for the Behavioral Sciences**. New York, McGraw Hill.

Silberman, E.K. & Weingartner, H. (1986), Hemispheric lateralization of functions related to emotion. **Brain and Cognition**, 5, 322-353.

Sokolov, E.N. (1963), **Perception and the Conditioned Reflex**. Oxford, Pergamon Press.

Skinner, B.F. (1985), Cognitive science and behaviourism. **British Journal of Psychology**, 76, 291-301.

Solomon, R.L. & Wynne, L.C. (1954), Traumatic avoidance learning: The principles of anxiety conservation and partial irreversibility. **Psychological Review**, 61, 353-385.

Stern, R.M., Ray, W.J. & Davis, C.M. (1980), **Psychophysiological Recording**. New York, Oxford University Press.

Stern, J.A., Walrath, L.C. & Goldstein, R. (1984), The endogenous eyeblink. **Psychophysiology**, 21, 22-33.

Sturgis, E.T. & Scott, R. (1984), Simple phobia. In S.M. Turner (Ed.). **Behavioral Theories and Treatment of Anxiety**. New York, Plenum Press, pp. 91-141.

Sulloway, F.J. (1979), **Freud, Biologist of the Mind. Beyond the Psychoanalytic Legend**. New York, Basic Books.

Tucker, D.M. (1981), Lateral brain function, emotion, and conceptualization. **Psychological Bulletin**, 89, 19-46.

Turner, S.M. & Michelson, L. (1984), Obsessive-compulsive disorders. In S.M. Turner (Ed.). **Behavioral Theories and Treatment of Anxiety**. New York, Plenum Press, pp. 239-277.

Valentine, C.W. (1930), The innate basis of fear. **Journal of Genetic Psychology**, 37, 394-419.

Vaitl, D., Gruppe, H. & Kimmel, H.D. (1985), Contextual stimulus control of conditional vasomotor and electrodermal reactions to angry and friendly faces; Transswitching - Yes! Preparedness - No! **Pavlovian Journal of Biological Science**, 20, 124-131.

Venables, P.H. & Christie, M.H. (1973), Mechanisms, instrumentation, recording techniques and quantification of responses. In W.F. Prokasy & D.C. Raskin (Eds.). **Electrodermal Activity in Psychological Research**. New York, Academic Press, pp. 1-124.

Vila, J. & Beech, H.R. (1977), Vulnerability and conditioning in relation to the human menstrual cycle. **British Journal of Social and Clinical Psychology**, 16, 69-75.

Watson, J.B. (1924), **Behaviorism**. New York, Norton.

Watson, J.B. & Rayner, R. (1920), Conditioned emotional reactions. **Journal of Experimental Psychology**, 3, 1-14.

Weber, S.L. & Sackheim, H.A. (1984), The development of functional brain asymmetry in the regulation of emotion. In N.A. Fox & R.J. Davidson (Eds.). **The Psychobiology of Affective Development**, Hillsdale NJ, Erlbaum, pp. 325-351.

Whittick, A. (1960), **Symbols, Signs, and Their Meaning**. London, Leonard Hill.

Wied, de D. & Jolles, J. (1982), Neuropeptides derived from pro-opiocortin: Behavioral, psychological, and neuro-chemical effects. **Physiological Reviews**, 62, 976-1060.

Wilde, G.J.S. (1962), **Neurotische labiliteit Gemeten Volgens de Vragenlijst Methode**. Amsterdam, Van Rossum.

Wilson, E.O. (1975), **Sociobiology: The New Synthesis**. Cambridge M, Harvard University Press.

Wilson, E.O. (1978), **On Human Nature**. Cambridge M, Harvard University Press.

Winer, B.J. (1962), **Statistical Principles in Experimental Design**. New York, McGraw-Hill.

Wittenmeyer, R., Stern, J.A. & Chen, S.C. (1983), Elevation of visual threshold associated with eyeblink onset. **International Journal of Neuroscience**, 18, 279-286.

Wolpe, J. (1981), The dichotomy between classical conditioned and cognitively learned anxiety. **Journal of Behavior Therapy and Experimental Psychiatry**, 12, 35-42.

Wolpe, J. & Lang, P.J. (1964), A fear survey schedule for use in behavior therapy. **Behaviour Research and Therapy**, 2, 27-30.

Wolpe, J., Lande, S.D., McNally, R.J. & Schotte, D. (1985), Differentiation between classically conditioned and cognitively based neurotic fears: Two pilot studies. **Journal of Behavior Therapy and Experimental Psychiatry**, 16, 287-293.

Wolpe, J. & Rachman, S. (1960), Psychoanalytic evidence: A critique based on Freud's case of little Hans. **Journal of Nervous and Mental Disease**. 131, 135-147.

Yerkes, R.M. & Yerkes, A.W. (1936), Nature and conditions of avoidance (fear) response in chimpanzee. **Journal of Comparative Psychology**, 21, 53-66.

Zafiropoulou, M. & McPherson, F.M. (1986), "Preparedness" and the severity and outcome of clinical phobias. **Behaviour Research and Therapy**, 24, 221-222.

Zung, W.W. (1965), A self-rating depression scale. **Archives of General Psychiatry**, 12, 63-70.

SAMENVATTING

Volgens Seligman's (1970) "preparedness"-hypothese is klassieke conditionering onderhevig aan evolutiebiologische invloeden. Seligman stelt dat CS-UCS associaties vooral aanleiding geven tot krachtige geconditioneerde responsen indien zij relevant zijn of waren voor de overlevingskansen van het organisme. Een dergelijk, door de evolutie gepotentieerd conditioneringsproces zou, aldus Seligman (1971), een sleutelrol spelen in de etiologie van fobische angsten. Het is deze bewering die in dit proefschrift op haar wetenschappelijke merites wordt beoordeeld.

Hoofdstuk 1 beschrijft de historische context waarbinnen men Seligman's hypothese moet plaatsen. Met name wordt gewezen op twee tendensen die zich in het begin van de zeventiger jaren begonnen af te tekenen. Ten eerste groeide het besef dat het klassieke conditioneringsmodel inzake fobieën ernstig tekort schoot. Ten tweede was er sprake van een toenemende belangstelling voor evolutiebiologische theorieën. Deze belangstelling hing zeker ook samen met de ontdekking dat conditionering onder bepaalde omstandigheden een uitgesproken selectieve vorm aanneemt. Een welhaast paradigmatisch voorbeeld hiervan biedt onderzoek waarin werd gevonden dat ratten zeer snel een verband tussen voedsel en misselijkheid leggen maar niet of uiterst traag voedsel met elektrocutane schokken associëren. Het ligt voor de hand te veronderstellen dat deze selectiviteit in natuurlijke situaties aanzet tot het vermijden van potentieel giftig voedsel en zo een bijdrage levert aan de overlevingskansen van het organisme. De essentie van de "preparedness"-hypothese wordt gevormd door de gedachte dat selectieve associaties ook ten grondslag liggen aan fobieën.

In hoofdstuk 2 wordt deze gedachte, zowel in Seligman's oorspronkelijke versie als in Öhman's meer recente versie, nader toegelicht. Seligman benadrukt dat het geen toeval is dat de meeste fobische angsten gericht zijn op een relatief kleine groep van objecten en situaties (engte ruimtes, hoogte, slangen etc.). Hij meent dat het hier gaat om stimuli die voor de prehistorische mens levensbedreigend waren. Het vermogen om met angst te reageren op deze stimuli zou onder invloed van natuurlijke selectiemechanismen zijn geraakt. En deze ontwikkeling zou op

haar beurt ertoe hebben geleid dat de moderne mens nog altijd is uitgerust met een genetische predispositie ("preparedness") om angst ten toon te spreiden ten opzichte van stimuli die ooit bedreigend waren. Volgens deze interpretatie zijn fobieën de uitkomst van een genetisch ondersteund conditioneringsproces dat de volgende eigenschappen bezit: het proces voltrekt zich snel, is selectief en resulteert in responsen die traag uitdoven en non-cognitief van aard zijn.

Aan de hand van moderne ethologische concepten ("closed genetic programs", "interspecific behavior" etc.) hebben Öhman en Dimberg (1984) een poging ondernomen om een meer gedetailleerde hypothese te formuleren over de wijze waarop bij fobieën genetische en leerfactoren in elkaar grijpen. Verder stellen zij dat niet alleen de enkelvoudige maar ook de sociale fobieën aan een "prepared" conditioneringsproces kunnen worden toegeschreven.

Hoofdstuk 3 biedt een overzicht van het empirisch onderzoek dat de "preparedness"-hypothese lijkt te ondersteunen. Aan de orde komen, achtereenvolgens, epidemiologische, klinische, experimenteel-psychologische en dierexperimentele studies. Voorstanders van de "preparedness"-hypothese verwijzen nogal eens naar epidemiologisch onderzoek waarin gevonden werd dat in normale populaties angst voor sommige stimuli (b.v. spinnen, slangen) aanmerkelijk vaker voorkomt dan angst voor andere stimuli (b.v. tandartsen, ziekenhuizen). Zij menen dat deze non-arbitraire verdeling een goede weerspiegeling is van de mate waarin stimuli een rol van belang in het overleven van de homo sapiens hebben gespeeld.

Ook diverse klinische studies lijken het idee te bevestigen dat de meeste fobische angsten gericht zijn op stimuli die bedreigend waren voor de prehistorische mens.

Achter het experimenteel onderzoek naar "preparedness" gaat de volgende redenering schuil: als fobieën berusten op een evolutionair verankerde predispositie dan moet in principe deze predispositie ook geactiveerd kunnen worden bij normale proefpersonen. Het experimenteel onderzoek valt uiteen in twee categorieën. De eerste categorie omvat de zogenaamde "illusory correlation" experimenten. Deze experimenten tonen aan dat indien fobie-relevante (b.v. spinnen) en neutrale stimuli (b.v. bloemen) even vaak samengaan met elektrische prikkels, proefpersonen toch

geneigd zijn om de contingentie tussen fobie-relevante stimuli en elektrische prikkels te overschatten. De tweede, en belangrijkste, categorie bestaat uit de psychofysiologische conditioneringsexperimenten van Öhman en medewerkers. Gebruikmakend van een elektrische prikkel als UCS vonden deze onderzoekers dat op fobie-relevante stimuli (slangen, spinnen, boze gezichten) geconditioneerde huidgeleidingsresponsen (SCRs) trager uitdoven dan op neutrale stimuli (paddestoelen, bloemen, neutrale gezichten) geconditioneerde SCRs. Voorts lieten zij zien dat één enkele associatie tussen stimulus en elektrische prikkel volstaat om dit soort differentiële uitdovingseffecten op te roepen. Tenslotte suggereren hun bevindingen dat de trage uitdoving van op fobie-relevante stimuli geconditioneerde SCRs niet ondermijnd wordt door expliciete "no more shock" instructies aan de proefpersonen. Al met al lijken deze bevindingen goed in overeenstemming te zijn met de eigenschappen die Seligman kenmerkend acht voor "prepared" leren. Öhman en medewerkers pretenderen dan ook dat het conditioneren van sympatische reacties op afbeeldingen van slangen (of spinnen) en van boze gezichten een geschikte, modelmatige reconstructie biedt van de etiologie van, respectievelijk, enkelvoudige en sociale fobieën.

De in dit verband relevante dierexperimenten werden uitgevoerd met rhesus apen. In het laboratorium opgegroeide apen koesteren geen angst voor slangen. Maar zodra deze apen soortgenoten observeren die terugdeinzen voor een slang ontwikkelen zij een sterke angst voor slangen. Het selectieve karakter van deze observationele conditionering blijkt uit het feit dat in het laboratorium opgegroeide apen geen angst voor, bijvoorbeeld, bloemen aan de dag leggen wanneer zij soortgenoten hebben gezien die van bloemen schrikken.

Hoofdstuk vier bestaat uit een zevental onderzoeken die werden verricht door de schrijver van dit proefschrift. Een telkens terugkerende constatering in dit hoofdstuk is dat allerlei bevindingen die in de literatuur ten faveure van de "preparedness"-hypothese worden opgevoerd niet repliceerbaar zijn. Zo worden in paragraaf 4.3 resultaten van een klinische studie gepresenteerd die regelrecht in strijd zijn met de bewering dat de meeste angsten betrekking hebben op voor de prehistorische mens bedreigende stimuli. In het in paragraaf 4.5 beschreven onderzoek werd tevergeefs geprobeerd de door Öhman gevonden trage extinctie van op

haar beurt ertoe hebben geleid dat de moderne mens nog altijd is uitgerust met een genetische predispositie ("preparedness") om angst ten toon te spreiden ten opzichte van stimuli die ooit bedreigend waren. Volgens deze interpretatie zijn fobieën de uitkomst van een genetisch ondersteund conditioneringsproces dat de volgende eigenschappen bezit: het proces voltrekt zich snel, is selectief en resulteert in responsen die traag uitdoven en non-cognitief van aard zijn.

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fobische stimuli geconditioneerde SCRs te valideren. Het in paragraaf 4.6 behandelde onderzoek toont aan dat de snelheid waarmee geconditioneerde SCRs uitdoven meer afhankelijk is van de wijze waarop proefpersonen worden geïnstrueerd dan van de stimulusinhoud (fobie-relevant vs. neutraal). Deze bevinding valt moeilijk te rijmen met het idee dat "prepared" leren non-cognitief van aard is.

Een tweede kwestie die in dit hoofdstuk veelvuldig naar voren komt betreft de validiteit van een aantal theoretische veronderstellingen waarop de "preparedness"-hypothese berust. In paragraaf 4.1 wordt bijvoorbeeld vastgesteld dat de angst voor fobie-relevante stimuli wel eens meer zou kunnen samenhangen met de perceptuele saillantie dan met de evolutionaire achtergrond van deze stimuli. De resultaten in paragraaf 4.2 maken duidelijk dat fobie-relevante en neutrale stimuli over het algemeen niet alleen van elkaar verschillen in termen van evolutionaire status, maar ook, en zelfs meer, in termen van (ontogenetische) gevaarlijkheid en onvoorspelbaarheid. In paragraaf 4.4 wordt verslag gedaan van een onderzoek dat geen enkele aanwijzing vond voor de veronderstelling dat bij sociaal fobici kwade gezichtsuitdrukkingen als fobie-relevante stimuli fungeren.

Op grond van de boven summier weergegeven onderzoeksresultaten lijkt het dringend gewenst om de theoretische uitgangspunten van de "preparedness"-hypothese aan een kritische analyse te onderwerpen. Zulks geschiedt in hoofdstuk 5. Er wordt vooral gewezen op het feit dat het de "preparedness"-hypothese ontbreekt aan heldere en uitgewerkte ideeën omtrent de genetische transmissie van angst.

In hoofdstuk 6 passeren een groot aantal studies de revue die, direct of indirect, de empirische kredietwaardigheid van de "preparedness"-hypothese ondergraven. Deze studies kunnen als volgt worden samengevat. Ten eerste blijkt bij een nadere beschouwing dat de epidemiologische onderbouwing van de "preparedness"-hypothese van een twijfelachtige kwaliteit is. Sommige onderzoeken mogen dan vinden dat angst voor slangen vaker voorkomt dan angst voor tandartsen, maar er is reden om te vermoeden dat het beperkt aantal items in deze onderzoeken mede-verantwoordelijk is voor dit resultaat. Zo is er een studie bekend waarbij de respondenten een groot aantal items kregen voorgelegd en waarin een angst-hiërarchie werd gevonden die zich moeilijk laat verenigen met de

"preparedness"-gedachte. In deze studie werd, onder andere, gevonden dat de angst voor God veel frequenter voorkomt dan angst voor slangen.

Op de tweede plaats kunnen de eerder aangehaalde klinische studies van de nodige kanttekeningen worden voorzien. De belangrijkste kanttekening is wel dat een fobie in de regel meerdere elementen (object van angst, vermijding, consequentie van vermijding) omvat. Sommige elementen zullen makkelijk en andere zullen moeilijk in evolutionaire termen te interpreteren zijn. Het is plausibel om te veronderstellen dat beoordelaars die een zekere affiniteit met de "preparedness"-hypothese hebben zich concentreren op de eerste groep van elementen en, langs deze weg, tot een overschatting van het aantal "evolutionair-relevante" fobieën komen.

Een derde punt betreft het "illusory correlation"-paradigma. De onderzoekers die van dit paradigma gebruik maken hebben tot nog toe verzuimd om in hun experimenten fobie-relevante stimuli met saillante en in objectieve zin gevaarlijke stimuli (b.v. geweren) te vergelijken. Het valt niet uit te sluiten dat zo'n vergelijking laat zien dat proefpersonen ook neigen tot het leggen van een illusionaire correlatie tussen stimulus en schok indien de stimulus saillant is en negatieve connotaties maar geen evolutionaire overlevingswaarde heeft. Daarmee zou het optreden van illusionaire correlaties niet specifiek zijn voor de zogenaamde fobie-relevante stimuli.

Tenslotte wordt uitvoerig ingegaan op het psychofysiologisch conditioneringsonderzoek naar "preparedness". Een zorgvuldige beschouwing van de literatuur leert dat van de zes pogingen om het door Öhman gerapporteerde differentiële uitdovingseffect te repliceren er maar liefs vijf op een totale mislukking zijn uitgelopen. Bovendien staat vast dat van de zeventien experimenten waarin geprobeerd werd dit effect onder iets andere condities op te wekken er tenminste elf faalden.

In hoofdstuk 7 wordt de balans opgemaakt. Een systematische evaluatie van de voorspellingen waaraan de voorstanders van de "preparedness"-hypothese zich wagen enerzijds en het onderzoeksmateriaal dat voorhanden is om deze voorspellingen te dekken anderzijds leidt tot de conclusie dat de "preparedness"-hypothese, empirisch gezien, zwak in haar schoenen staat. Terwijl evolutiebiologische theorieën in het algemeen en de "preparedness"-hypothese in het bijzonder onderstrepen dat elke soort een

specifieke ecologische niche heeft en comparatieve studies derhalve misleidend kunnen zijn, biedt, ironisch genoeg, het dierexperimentele onderzoek de meest krachtige ondersteuning aan de "preparedness"-hypothese. Waar het gaat om de etiologie van humane fobieën kan de "preparedness"-hypothese niet langer als veelbelovend gelden.

Het een en ander houdt evenwel niet in dat daarmee ook conditioneringsmodellen inzake fobieën aan waarde hebben ingeboet. Er wordt uitdrukkelijk vastgesteld dat de moderne conditioneringsliteratuur interessante aanknopingspunten biedt voor het ontwikkelen van een leertheoretische visie op het ontstaan van fobieën. Daar dient echter aan toegevoegd te worden dat zo'n visie waarschijnlijk meer baat heeft bij inzichten uit de neurowetenschappen dan bij evolutionaire speculaties.

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LIST OF PUBLICATIONS AND ABSTRACTS

1.* Bögels, S. & Merckelbach, H. (1988), Buitenlanders behandelen met biofeedback. **Maandblad Geestelijke Volksgezondheid, 43**, 174-175.

2. Cohen, L. & Merckelbach, H. (1987), Dichotic listening in relation to dysphoria and other personality characteristics. **Perceptual and Motor Skills, 64**, 471-477.

3. Hoekstra, R. & Merckelbach, H. (1987), Dwangneurosen: Geen verklaring; Wel therapie. **Intermediair, 23**, 45-49.

4. Hout, van den M.A., Merckelbach, H., Hoekstra, R. & Oosterlaan, J. (1989), Is washing a prepared ritual? **Scandinavian Journal of Behaviour Therapy**, in press.

5. Hout, van den M.A., Zijlstra, B. & Merckelbach, H. (1989), Selective recall of surprising visual scenes: An experimental note on Seligman and Yellen's theory of dreams. **Behaviour Research and Therapy**, in press.

6. Jansen, A., Klaver, J., Merckelbach, H. & Hout, van den M.A. (1989), Restrained eaters are rapidly habituating sensation seekers. **Behaviour Research and Therapy**, in press.

7. Jansen, A. & Merckelbach, H. (1988), De wens is de vader van de gedachte: Drie beroemde gevalsbeschrijvingen geanalyseerd. **De Psycholoog, 23**, 41-44.

8. Jansen, A., Merckelbach, H., Hout, van den M.A. & Griez, E. (1988), Over-eten, koolhydraten en serotonine. **Nederlands Tijdschrift voor de Psychologie, 43**, 159-167.

9. Jansen, A., Merckelbach, H., Oosterlaan, J., Tuiten, A. & Hout, van den M.A. (1988), Cognitions and self-talk during food intake of restrained and unrestrained eaters. **Behaviour Research and Therapy, 26**, 393-398.

10. Jansen, A., Oosterlaan, J., Merckelbach, H. & Hout, van den M.A (1988), Non-regulation of food intake in restrained, emotional, and external eaters. **Journal of Psychopathology and Behavioral Assessment, 10**, 345-353.

11. Merckelbach, H. (1985), De fantasie van het gespleten brein. **Intermediair, 21**, 29-33.

12. Merckelbach, H. (1986), Zijn fobieën een evolutionaire last? **Psychologie, 5**, 14-17.

13.* Merckelbach, H. (1987), Bespreking van "Topics in behavioral medicine" (Vinck, Vandereycken, Fontaine & Eelen; Eds.). **Gedragstherapie, 20**, 231-232.

14.* Merckelbach, H. (1988), Bespreking van "Cognitive processes and Pavlovian conditioning in humans" (Davey; Ed.). **Gedragstherapie, 21**, 75-77.

15.* Merckelbach, H. (1988), Geconditioneerde inhibitie van sympathische reacties op pijnstimuli in een aversieve conditioneringsprocedure, **De Psycholoog, 23**, 460.

16. Merckelbach, H. (1988), Endogene oogknipperingen: Angst of aandacht? **De Psycholoog, 23**, 659-662.

17.* Merckelbach, H. (1988), Bespreking van "Panic and phobias 2" (Hand & Wittchen; Eds.). **Gedragstherapie, 22**, 63-65.

18.* Merckelbach, H. & Hout, van den M.A. (1987), Phobias and preparedness: Disappointing results from four studies. **Psychophysiology, 24**, 566-567.

19. Merckelbach, H. & Hout, van den M.A. (1988), Electrodermal and cardiovascular responses to phobia-relevant stimuli in a trace conditioning paradigm: Effects of instruction. **Journal of Psychophysiology**, 2, 181-193 (section 4.6).

20. Merckelbach, H. & Hout, van den M.A. (1989), Fear relevance and diminution of unconditioned skin conductance responses, submitted (section 4.7).

21. Merckelbach, H., Hout, van den M.A., Hoekstra, R. & Oppen, van P. (1988), Are prepared fears less severe, but more resistant to treatment? **Behaviour Research and Therapy**, **26**, 527-530 (section 4.3).

22. Merckelbach, H., Hout, W., Hout, van den M.A. & Mersch, P.P. (1989), Psychophysiological and subjective reactions to facial stimuli in social phobics and normals. **Behaviour Research and Therapy**, in press, (section 4.4).

23. Merckelbach, H., Hout, van den M.A., Jansen, A., & Molen, van der G.M. (1988), Many stimuli are frightening but some are more frightening than others: The contributions of preparedness, dangerousness, and unpredictability to making a stimulus fearful. **Journal of Psychopathology and Behavioral Assessment, 10**, 355–366 (section 4.2).

24. Merckelbach, H., Hout, van den M.A., & Molen, van der G.M. (1987a), Preparedness en fobieën: Een kritisch literatuuroverzicht. **Nederlands Tijdschrift voor de Psychologie, 42**, 115–124.

25. Merckelbach, H., Hout, van den M.A. & Molen, van der G.M. (1987b), Fear of animals: Correlations between fear ratings and perceived characteristics. **Psychological Reports, 60**, 1203-1209 (section 4.1).

26. Merckelbach, H., Hout, van den M.A. & Molen, van der G.M. (1989), The phylogenetic origin of phobias: A review of the evidence. In Emmelkamp, P.M.G. (Ed.). **Three perspectives on Anxiety Disorders: Biological, Behavioral, and Cognitive Models.** Amsterdam, Swets, in press.

27. Merckelbach, H., Hout, van den M.A. & Oppen, van P. (1988), Failure to condition evaluative and electrodemal responses to neutral stimuli by means of prepared cues. **Activitas Nervosa Superior, 30**, 161-167.

28. Merckelbach, H. & Jansen, A. (1986), Emotie, cognitie en het ontstaan van fobieën. **Gedragstherapie, 19**, 145-160.

29. Merckelbach, H., Jansen, A., Hout, van den M.A., & Molen, van der G.M. (1988), Vasomotorische reacties: Zwakke conditionering en geen "preparedness". **Gedragstherapie, 21**, 3-14.

30.* Merckelbach, H. & Jansen, A. (1989), Bespreking van "Theoretical foundations of behavior therapy" (Eysenck & Martin; Eds.). **Gedragstherapie, 22**, 73-75.

31. Merckelbach, H., & Jong, de P. (1988), Geconditioneerde inhibitie van psychofysiologische reacties op pijnprikkels: Feiten en speculaties. **Psychologica Belgica, 18**, 139-155.

32. Merckelbach, H., & Jong, de P. (1989), Conjugate lateral eye movements, cerebral dominance, and anxiety. Submitted.

33. Merckelbach, H., Molen, van der G.M. & Hout, van den M.A. (1987), Electrodermal conditioning to stimuli of evolutionary significance: Failure to replicate the preparedness effect. **Journal of Psychopathology and Behavioral Assessment**, 9, 313-326 (section 4.5).

34. Merckelbach, H. & Oppen, van P. (1989), Effects of gaze manipulation on subjective evaluation of neutral and phobia-relevant stimuli: A comment on Drake. **Acta Psychologica**, 70, 1-5.

35. Merckelbach, H., Ruiter, de C. & Olff, M. (1989), Handedness and fear in normal and clinical populations. Submitted.

36. Molen, van der G.M., Merckelbach, H. & Hout, van den M.A. (1988), The possible relation of the menstrual cycle to susceptibility to fear acquisition. **Journal of Behavior Therapy and Experimental Psychiatry**, 19, 127-133.

37. Molen, van der G.M., Hout, van den M.A., Merckelbach, H., Dieren, van A.C. & Griez, E. (1989), The effect of hypocapnia on extinction of conditioned fear responses. **Behaviour Research and Therapy**, in press.

38*. Zijlstra, B.C., Merckelbach, H. & Hout, van den M.A. (1988), Hemisfere dominantie en het Von Restorff-effect. **De Psycholoog**, 23, 509.

* Abstract or Book Review.

CURRICULUM VITAE

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